Exhibit A

negative tTG at time of that initial consideration of celiac disease can be helpful. But, again, the diagnosis is based on the response to olmesartan withdrawal, not the results of the celiac blood test.

Q. Doctor, you understand and agree that celiac disease is something different than sprue-like enteropathy associated with olmesartan?

A. Yes, these are different conditions.

Q. And you agree that olmesartan does not cause celiac disease, correct?

A. So olmesartan does not cause -- we do not believe causes celiac disease.

There have been some cases reported where patients after having olmesartan -- what appears to be olmesartan enteropathy are then later found to also have celiac disease. Whether or not an initial injury to the small intestine causing increased passage of antigens, such as gluten, could in some cases trigger celiac disease in an otherwise genetically predisposed individual I think is an open question. It's plausible. We see that with other conditions.

We see patients develop celiac disease after neurovirus, a small intestinal infection. So we know small intestinal injury can lead to autoimmune diseases like celiac disease, so there are different conditions. And in most cases, people with olmesartan enteropathy once they stop the olmesartan will be fine, but there have been reports of cases who appear to develop celiac disease afterwards, and that would be consistent.

Q. Objection, non-responsive starting with "there have been" to the end of the answer.

Dr. Leffler, you have never diagnosed someone with what you call olmesartan enteropathy and then subsequently found that they had celiac disease that was caused from the olmesartan enteropathy?

A. In my practice, I have not.

Q. When did you first yourself learn about sprue-like enteropathy?

MS. SUTTON: Objection, form.

A. Was this referring to olmesartan enteropathy, sprue-like enteropathy associated with olmesartan?

Q. I wanted to be clear, that's why I started off with our definition. When I ask you

the most frequently used cell lines in gastroenterology to look at intestinal previability and inflammation.

Q. Both large and small intestine?

A. Both large and small intestine. They actually induce features very similar to many aspects of the small intestine. Actually, one of the people you referenced earlier, Jerry Turner, is one of the leaders in this area and has done a lot of work on looking at Caco-2 lines and how they apply and have relevance to small intestinal diseases.

Q. So for the Caco-2 cell line testing, do you know what dosage of olmesartan they used in that in vitro testing?

A. So let me see. In Figure 8 I can see that they report 30 micromole per liter. Again, it's hard to -- we -- so let me clarify.

So it's not entirely clear what millimole level the intestine actually sees of olmesartan. We can't compare systemic levels of olmesartan to what the intestine sees when the pill is there and it's absorbing.

The levels that the intestine sees of any drug are by definition often much, much

Exhibit B

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY

IN RE: BENICAR (OLMESARTAN)
PRODUCTS LIABILITY LITIGATION

CIVIL ACTION NUMBER:

15-2606

Mitchell H. Cohen United States Courthouse
One John F. Gerry Plaza
Camden, New Jersey 08101
January 25, 2017

B E F O R E: THE HONORABLE ROBERT B. KUGLER
UNITED STATES DISTRICT JUDGE
UNITED STATES MAGISTRATE JUDGE JOEL SCHNEIDER

A P P E A R A N C E S:

ADAM SLATER, ESQUIRE
ATTORNEY FOR PLAINTIFFS

RICHARD GOLOMB, ESQUIRE
ATTORNEY FOR PLAINTIFFS

STEVEN RESNIK, ESQUIRE
ATTORNEY FOR PLAINTIFFS

RAYNA KESSLER, ESQUIRE
ATTORNEY FOR PLAINTIFFS

SUSAN M. SHARKO, ESQUIRE
ATTORNEY FOR DEFENDANTS
MICHAEL C. ZOGBY, ESQUIRE
ATTORNEY FOR DEFENDANTS

KENNETH A. MURPHY, ESQUIRE
ATTORNEY FOR DEFENDANTS

JESSICA BRENNAN, ESQUIRE
ATTORNEY FOR DEFENDANTS

AMY KLUG, ESQUIRE
ATTORNEY FOR DEFENDANTS

Certified as true and correct as required by Title 28, U.S.C., Section 753.

/S/ Carl J. Nami

(open court)

THE DEPUTY COURT CLERK: All rise.

THE COURT: Thanks. Have a seat. All right. Good afternoon. How is everybody?

00:20 All right. Have a seat. Relax. Where is our man from New Orleans?

MR. SLATER: He said he had enough of these conferences because nothing ever happens.

THE COURT: Since he's not here, we're going to 00:20 appoint him to something that he's not going to like. How about that?

MR. SLATER: I think that, that -- he should be on the lunch committee. He had this deposition and he said he couldn't get out of it.

00:21 THE COURT: That's fine. No big deal. All right. Welcome back, everybody. Just one preliminary thing I want to bring to everybody's attention. Defense counsel already knows this. We are trying a to construct, for lack of a better term, a spread sheet for the court so we can keep track of all 00:21 this stuff, and one data point that we don't have, so we've asked defense counsel if they could help us with this. We want to put in our spread sheet a very general description of the injuries being claimed by each of the separate plaintiffs. So, they've offered to give us just a very short description 00:21 of the plaintiffs' injuries so we can put it in those spread

sheets so we can track of who's doing what. Okay?

MR. SLATER: Just for the Bell Weather cases?

THE COURT: No. It's going to be --

MR. SLATER: Cross litigation?

00:22 MS. SHARKO: And that will come from the PFS, not from medical records.

THE COURT: Right, it comes from the PFS. We don't have access to the PFS. I'm not asking that we have access to the PFS.

00:22 MR. SLATER: We can have them delivered in hard copy.

THE COURT: Well, thank you. That I don't need at this point.

All right, I have your proposed agenda. I have a number of complaints in the Federal and State litigation. We

00:22 have first time listing on page one, paragraph three. That's still on?

MS. SHARKO: Yes. No changes there.

THE COURT: Okay. The second time listing there's two of them listed?

00:22 MS. SHARKO: Yes. No changes. They should be allotted to Orders to Show Cause.

MR. GOLOMB: Your Honor, on the Mason case. That's my case.

THE COURT: Yes.

00:22 MR. GOLOMB: And we're asking for another 30 days

there. Mason does not recall the name of his physician but remembers the clinic that he went to and that clinic is closed down. So we're trying to identify where they forwarded the records to and we just request another 30 days before this Order.

00:23

THE COURT: Well, you're going to get the 30 days because that's how long it's going to take for the Order to Show Cause to be returnable anyway, and then if you have more information, then at the next status conference I want you to tell me about it.

00:23

MR. GOLOMB: Okay.

THE COURT: So that we don't dismiss it. I'll listen to you then.

MR. GOLOMB: Okay.

00:23

THE COURT: Then we'll see what happens.

MR. GOLOMB: Okay. Thank you.

THE COURT: Paragraph four, overdue fact sheets, first time listing.

MS. SHARKO: We are down to 37.

00:23

THE COURT: Down to 37.

MS. SHARKO: So number four Branch.

THE COURT: Is out?

MS. SHARKO: That comes off the list. Number nine, Curiton. C-u-r-i-t-o-n, is off. They served a PFS yesterday.

00:24

Number 24, Moore is off. They served a PFS yesterday.

Number 26, Plater. P-l-a-t-e-r, is off. They served a PFS yesterday.

Number 28, Rhymes as in nursery. They're off. They served a PFS yesterday.

00:24 Number 40, Wells is off. They served the PFS yesterday. And number 41, Westbrook, is off. They served a PFS yesterday.

MR. GOLOMB: Your Honor, number 27 should come off as well. That's Roland. We've already filed a stipulation of

00:24 dismissal.

THE COURT: Okay.

MS. SHARKO: I don't know one way or the other, but we'll watch for it.

THE COURT: We'll obviously take counsel's word for

00:25 it. Well, you know, at this rate I think we calculated with these stipulations of dismissal it will be 2037 we'll have all these cases dismissed? Justice at work.

All right, second time listing.

MS. SHARKO: Second time listed all nine cases are

00:25 still deficient and should go on to an Order to Show Cause, please.

MR. SLATER: One second, Your Honor. Case four on that list, Hoker. I've been asked by counsel from Wagstaff and Carmel to make an application to the court for a 90 day

00:25 extension. The plaintiff died recently. So the court -- so

they asked for an extension to have time for the family to have a representative appointed et cetera and to take care of all that and apparently from the defense would not consent to an extension. So, we're asking the Court if we could have an
00:25 extension for someone who just died.

MS. SHARKO: And so here's our position. We got that request yesterday. This case was filed on June 30th of last year. It was served on July 8th. The PFS was due on October 6th. We got nothing. An overdue letter was sent on
00:26 October 14th. We got nothing. It was listed on the November agenda as a first timer. We got nothing. And then now it's a second time. Apparently according to out internet research, the man died in December. So, our sympathies to the family, but we have nothing on this case at all.

00:26 THE COURT: We're going to list it again on the second listing. So that we'll give them another 30 days and I want a report.

MS. SHARKO: Okay.

THE COURT: As to what steps have been taken to
00:26 substitute an Estate, if any, if they still want to continue.

MR. SLATER: To make sure they're actively doing it.

THE COURT: Yes. Okay?

MR. SLATER: Okay. I know they do intend to and they actually I can tell you, they told me that a PFS was basically
00:26 filled out. They just don't know whether to sign it right now

because they don't have a rep. Thank you, Judge.

THE COURT: You got some more time.

MR. SLATER: Okay.

MS. SHARKO: We, frankly, would be more sympathetic
00:26 if we had a PFS that at least showed there was information there I guess that it takes a while to get an Estate in place.

THE COURT: All right. It takes a little while. We'll see.

MS. SHARKO: Okay.

00:27 THE COURT: Mr. Golomb.

MR. GOLOMB: Two cases on that list, Your Honor, Ballard, we have a similar situation. Mr. Ballard recently passed away and we're working with the surviving spouse to complete the fact sheet.

00:27 THE COURT: That's number one on the list. Correct?

MR. GOLOMB: Yeah.

MS. SHARKO: That's totally new to us. That case has now been around for several months. We have no information on it. I have no idea what the date of death is.

00:27 THE COURT: We'll give it another second listing.

MR. GOLOMB: And on Vickie Gains, we've already filed a stipulation of dismissal on that.

THE COURT: Okay.

MS. SHARKO: Okay. We look forward to seeing that.

00:27 (Brief pause)

THE COURT: Paragraph six, page 10. Two cases listed.

MS SHARKO: Yes, sir. So these are case where when you read the PFS, the injuries that are alleged come before 00:28 Benicar use, not after. And we've raised that question with plaintiffs. We've gotten no response, and we submit that those cases should be dismissed because usually it has to be taken within the then happened event.

THE COURT: Usually. What are we doing with these 00:28 two?

MR. SLATER: You know, our position is that, and obviously I don't have the medical records in front of me, but I think counsel is essentially looking for a expedited way to get Summary Judgment on a case. I think if when the time 00:28 comes, they can deal with it, but I don't have the medical facts in front of me. Maybe the person with gastrointestinal issues before and their experts are going to say, well, that was related to one thing and then the Olmesartan caused issues when they went on the drug, that's different and it would have 00:28 been more severe or if this wouldn't have happened. I mean that's all in the realm of very possible. I don't have the records to say I know the answer. But for them to pick out records and say they've analyzed it so the plaintiff has to prove their case at this point when it's not even a Bell 00:29 Weather case. I think it's not an issue for us to be doing

that now.

THE COURT: Well, we're going to deal with that issue by picking off the strategy in a few seconds. But I think when there's a specific request from defense counsel alerting
00:29 the plaintiffs that there is a specific problem, I think I need a little bit better response than that. Whoever represents these two people should be at least on the phone with Miss Sharko saying, well, this is what we think is going on here.

00:29 MR. SLATER: Okay.

THE COURT: Try to resolve this because if this is true, then Rule 11 would seem to indicate they shouldn't be continuing in these cases.

MR. SLATER: I'll talk to -- well, I know one is Mr.
00:29 Golomb's. So I certainly think that I can hand that off to him and on the other firm I'll talk to the other law firm and tell them they need to provide an explanation for why they think the case is a valid case.

THE COURT: Right.

00:30 MR. SLATER: Okay.

MS. SHARKO: We appreciate that. We sent letters out in mid November.

THE COURT: Maybe Mr. Golomb can give us an answer on the Hiddleston case.

00:30 MR. GOLOMB: I can, Your Honor. They're wrong on the

facts, but with that said, we're dismissing the case for other reasons.

THE COURT: You're going to file a stipulation?

MR. GOLOMB: Yes.

00:30 THE COURT: Okay. So get a response from Matthews and Associates to Miss Sharko, please, on that issue and open up some line of communication with Miss Sharko on this case.

MR. SLATER: Yes, I will.

THE COURT: Thank you. All right, now we get to

00:30 number seven which is the 82 cases which is now 80 cases?

MS. SHARKO: Right. Actually since we sent the letter to Your Honor, we've heard from eight plaintiffs. Two took dismissals with prejudice as a result of the letter I guess confirming we were right and six of the plaintiffs sent

00:31 records documenting an event and a prescription. So they would come off the list and we heard nothing from anyone else. I think the fact at least six people served records, shows that plaintiffs are holding records and not updating their PFS's. So we would ask that that be done.

00:31 THE COURT: Well, really it shows that you, plaintiff's counsel, if they have such records, were deficient in providing them to you. This is a problem with these stragglers. We will put into place an efficient method to dispose of the stragglers without you having to file summary

00:31 judgment in each and every one. So you're not going to handle

80 or a hundred summary judgment motions on these kind of cases. But I don't think this is the appropriate time to do it. I promise you we will get to that and you'll have a method by which to get rid of these cases. I want to focus
00:32 really more, all of your energies on the experts in getting that lined up for Daubert Hearings at this point. Okay?

MS. SHARKO: Okay.

THE COURT: All right, that's paragraph eight which is the defendant served its list of experts.

00:32 MS. SHARKO: We do and we have one addition to make to that. I was told this morning that there's one additional regulatory expert Dr. Feigal, F-e-i-g-a-l. And his deposition date has already been set some weeks ago. It was on a letter to plaintiffs and it was -- we didn't have it when we sent
00:32 this letter. So we apologize but that's -- he should have been on the list to Mr. Slater and to the court yesterday afternoon.

MR. SLATER: I don't know what, what -- we didn't accept any of the dates yet. So I'm not sure what date he's
00:33 on for, but we can sort that out and you can let us know. We certainly still have a significant issue here from our perspective, Judge.

THE COURT: We're going to deal with it right now. We'll talk about it right now.

00:33 MR. SLATER: Okay.

THE COURT: All these experts out there, that the plaintiff had a significant number of experts and the defendants have an extraordinary number of experts at this point.

00:33 Let me take you back to November 30th, and November 30th we had a conference, phone conference and at that point we were discussing, Mr. Slater was discussing the experts and I stated to you all on the phone, well, of course, recognized there's just no chance that you're going to be able to put 21 00:34 experts on the stand during the trial. And perhaps I was being too subtle, although I'm not usually accused of being too subtle. Folks, we are not going to entertain testimony from 50 experts and I'm not going to hear 50 Daubert motions regarding experts. It just isn't going to happen, and the 00:34 jury is never going to hear this number of experts. It's wasting time. So we're going to pare this down, and the first way we're going to pare this down is I want to concentrate, focus back to where I've always been since the beginning of the case. I want to focus first on the general causation 00:34 experts. Let's focus and get this done. Let's get the Daubert hearings done on them first. And, perhaps, I should have been clearer about that two months ago, but -- and that's my fault and I'm sorry. But we can still fix this problem. But I -- there are, as I count them now, 13 or 14 defense 00:35 general liability experts and the best I can tell from the

plaintiffs there are six. Correct?

MR. SLATER: That's correct, Your Honor.

THE COURT: Correct?

MS. SHARKO: Yes.

00:35 THE COURT: All right.

MS. SHARKO: But.

THE COURT: But?

MS. SHARKO: We have no intention of calling all of these experts in every case should we get to trial, but we're 00:35 mindful of the fact that we have ten plaintiffs and we have general and specific causation as to each of those ten plaintiffs. And so we didn't want to be in a position where we identify one expert and then he or she is not available for whatever reason when trial is set. So, number one, we don't 00:35 intend to call all those experts in any one trial. We know that you would never let us do that.

The second thing is that Daubert and general causation and specific causation cover a wide range of areas. And so we need people in all these different areas to address those 00:36 areas. And the third issue is simply one of scheduling. We don't know when the Daubert hearings are going to be. And, so, yes, we have a couple epidemiology experts.

THE COURT: Four. You have four.

MS. SHARKO: Right.

00:36 THE COURT: I suggest you don't need four.

MS. SHARKO: If we knew when the hearings were going to be, we could narrow that group. If we knew when the hearings were going to be and there was an agreement that if the cases were tried, we could use any of those four. We'd be
00:36 willing to narrow it to two.

Part of our concern is looking down the road at the big picture and making sure that we're not foreclosed.

THE COUR: Explain to me how the date of the hearing determines the number of experts that you're going to use.

00:37 MS. SHARKO: Okay. So, why, why did we name four. Because we're looking at these experts as trial experts in addition to the Daubert Hearings and then if you said no we could only have two, we need to make sure that those two, at least one of them is available to testify live at the Daubert
00:37 Hearing.

MAGISTRATE JUDGE SCHNEIDER: So do you anticipate at the Daubert Hearing separate rulings have to be made as to each of the four epidemiologists?

MS. SHARKO: It depends on what plaintiffs'
00:37 challenges are, and really if this is premature in the sense that they should see the reports. Another way to narrow this and I've been thinking about this because I'm sensitive to the issues you raised, if we have ten cases, we're not sure about all the dispositive motions we're going to make, but we know
00:38 that we're going to make statute of limitations motions in

two cases. If we took those two cases off this list and deferred discovery, expert discovery on that, that would get rid of a number of experts for both sides.

THE COURT: I don't think it's going to have a
00:38 significant impact on the number of experts on both sides. If you focus on general liability, general causation, does Olmesartan cause spruelike enteropathy, et cetera, et cetera and all the other maladies. Which of these experts are going to say yes or no?

00:38 MS. SHARKO: Well, Mr. Parker will answer that specifically as to the experts. But I don't think we can or should address General Causation in a vacuum. I think we should be doing general and specific causation in the context of the specific cases.

00:38 THE COURT: Why not in a vacuum? That's the full genesis of this case was that the plaintiffs felt that olmesartan causes these conditions and that's how they start and the manufacturer said no it doesn't. Until we get an answer to that question, where do we go?

00:39 MS. SHARKO: Well, the question really is do the plaintiffs' expert's opinion -- are the plaintiffs' expert opinions as to General Causation supported by sound scientific evidence.

THE COURT: Right.

00:39 MS. SHARKO: And we think that should be examined in

conjunction with a specific case.

THE COURT: How does it help me to examine it in conjunction with a specific plaintiff in a specific plaintiff's complaints of injury?

00:39 MS. SHARKO: Because if you don't, you're, you're adjudicating a question in a vacuum. It's an issue that's not anchored in the facts of one specific case.

THE COURT: It's anchored in the facts in every one of these 2000 cases. Every one of these 2000 cases is

00:40 dependent on the answer to that first question. If the answer is no, that's it. If the answer is yes, then they got to prove what their injuries are period. That's all it is.

So, I mean I know you've stated this before about the vacuum, but in essence it is a vacuum because of the way the

00:40 case is framed and the way the case was filed and the reason it's here. That the plaintiffs want an opportunity to prove what in effect is new science. And, you know, Daubert sets out how you do that. It's not an easy thing to do, but it's doable under Daubert. And it seems to me that we're wasting a

00:40 lot -- not wasting, but we're spending an awfully lot of time and effort and expense on both sides on everything else without attacking that crucial question yes or no.

MS. SHARKO: I think we are, and we're armed and ready to attack that, plus the specific causation in a given

00:41 case. We have ten cases on both sides rolled up and ready to

go as of January 31st when we serve our expert reports. To save time we could take out the two statute of limitations cases. That would give us eight cases, or Your Honor could randomly pick five of the ten. I think it should be random. 00:41 And then that would cut the number of experts down. But we believe that we should address specific and general causation simultaneously.

THE COURT: Mr. Slater? Mr. Golomb?

MR. SLATER: Well, obviously you know our position 00:42 which is they need to drastically cut the number of experts. I mean --

THE COURT: We're going to do that.

MR. SLATER: Okay. So -- and I think Your Honor's going to need information of what some of these people are 00:42 actually generally going to say and I think that's obvious to the court.

As far as cutting cases, I've never heard that until right now. So, I'd have to, I'd have to know what they're talking about in terms of which cases, why, what the benefit 00:42 -- what's the procedure later and I'd have to talk to a few other people. I didn't want unilaterally to make that call for other people who have a lot vested in this litigation and not that I'm afraid to make a decision but I think it's the appropriate thing to do. And I don't even know if the court 00:42 is inclined to do that. If Your Honor is, I can speak to

people, we can talk about it. But, you know, I'm in full agreement with the court that we should be focusing on what do we need to put together to prove yes or no that the drug causes this issue. And that's really the primary question.

00:42 And we've said, I've been in front of you many times saying I don't think when push comes to shove that they are going to oppose general causation. We'll see what happens with these reports and these depositions. I don't know, frankly, how they can do it when every scientific article in the literature

00:43 says it does. So I don't know how they oppose it under Daubert. I mean, frankly, they are going to put us to our test and whoever does testify, I would expect we're going to have motions back at them for having to give opinions without being rooted in the actual peer review literatures. I mean

00:43 there are some issues on the general causation that will keep the court quite busy and keep us busy anyway at the core of what the case is. So I think we have to stick to where we were which is they need to drastically cut down and we have to figure out who really needs to be deposed and which experts

00:43 really need to be in the case at this stage. If the court has any inclination to discuss paring the cases, I think we have to really understand what the court's inclined to do, what's being offered. How does it happen so I can talk with my team and give you a reason to respond to that. It's not just my

00:43 view but allows others that have an important investment in

this also to say something.

THE COURT: Well paring the cases, Miss Sharko says there's two of the ten in the statute of limitations issues. I mean that's rather straightforward.

00:44 MR. SLATER: And I don't know which cases those are.

THE COURT: Well, I don't either, but that was the point I wanted to make is that that may be something that the plaintiffs should discuss with Miss Sharko as to those two cases, and if she's right, then what's the point?

00:44 MR. SLATER: If she'll tell us which those are, we'll take a look at them.

THE COURT: I think you really ought to have a sit down, both sides as to those two cases because she even needs to file those kinds of motions I mean.

00:44 MR. SLATER: I understand that.

THE COURT: Those kinds of cases have been on a long time.

MAGISTRATE JUDGE SCHNEIDER: I thought the question, Mr. Slater, was whether in the first instance you want to go

00:44 to a Daubert trial on general causation or as Mr. Sharko is suggesting, general and specific causation.

MR. SLATER: Okay. Well, on that issue, Your Honor, when Judge Kugler raised that on that at that conference, we think it makes absolute sense to have a trial on a single

00:45 issue first because then the efficiency going forward is

compounded. I mean at that point if we prevail on that, these experts aren't coming in. You don't have to bring in an epidemiologist. You don't -- even if they're needed, which is -- you know, we'll see what happens. We were very careful to
00:45 make sure we covered our bases, so we win the basketball game against our epidemiologists. But they're good players. But, no, absolutely that from our perspective makes perfect sense to focus on getting to a trial in general causation first because for reasons the court has identified. Absolutely,
00:45 we're all for that and I think it makes a lot of sense and we're willing to try that, that case whenever the court tells us to.

MS. SHARKO: And we are opposed to that. We think that's a very significant due process. We're trying one issue
00:45 in a vacuum.

THE COURT: Well, it's been done. It's been affirmed by the Court of Appeals, and I'm strongly leaning in that direction, but before we get there. Let's talk about the experts. I want to focus on the first tranche of these
00:46 motions are going to be directed to the general liability experts. Each side is going to pick five general liability experts. Five. That's all you're going to have on your liability.

MR. SLATER: Do you mean on causation?

00:46 THE COURT: General causation.

MR. SLATER: Okay. I'm sorry.

THE COURT: I'm sorry. That's correct. Need to be more specific. And your Daubert motions are going to be directed at the five that your advisory has served on you. So
00:46 you're going to need to identify, Miss Sharko, when you serve those reports next week. Right?

MS. SHARKO: Yes, sir.

THE COURT: Which five you're going to be relying for general causation. You're going to have to notify her by the
00:46 end of that date the five that you're going to be relying on for general causation. Okay? So, by next week we're going to have ten experts going, there's going to be ten reports done, take the ten depositions, file the motions immediately, and we'll hear those immediately, and then we'll back fill in the
00:47 specific causation experts after that. Let's get these done first. It's lot of work. I want to focus on getting it done because that to me has always been the key of this case.

MS. SHARKO: Judge, we have experts in six different areas.

00:47 THE COURT: I know. So do they.

MS. SHARKO: They have experts in five areas and we would really need --

THE COURT: Which area is additional for you that they don't have? Why do you need an expert to testify in an
00:47 area they're not raising?

MS. SHARKO: Because we believe that each of the areas we have are important to the determination of the issue of General Causation. So we have pharmaco-vigilance.

THE COURT: Right.

00:47 MS. SHARKO: And they don't have anybody broken out separately as to pharmaco-vigilance. So we would ask for six on the variance, otherwise we have no one to speak to an area.

THE COURT: It's an area that they haven't raised.

MS. SHARKO: They have.

00:48 THE COURT: How have they raised the pharamco-vigilance?

MS. SHARKO: They have pharamco-vigilance addressed by their regulatory people and by their epidemiologists, and I think by their GI person and we have people who have specific

00:48 expertise in each of these six areas. And so if we cut out, if we're forced to cut out one of these and that's an area, that's a gap for us.

THE COURT: I just don't remember -- I've read their expert reports a long time ago. I just don't remember that

00:48 specific part of them.

MS. SHARKO: So they have two GI doctors. We have three. They have one pathologist. We have one general pathologist. They have one regulatory. We have two regulatories. They have one toxicologist. We have two

00:49 toxicologists. And then they have one epidemiologist and we

have four. And then we have two people whose specific area of expertise is pharmaco-vigilance. They absolutely raised pharamco-vigilance. That's one of the key issues in the case as Judge Schneider has told us over and over. But they
00:49 addressed pharmaco-vigilance but by having multiple of their experts talk about it and we broke it out and got people whose specific area of expertise that was to address that. So we would ask permission to have one of those.

THE COURT: All right. Mr. Slater, anything you want
00:49 to say?

MR. SLATER: Yeah. What I would say is I think, look, whether it's five experts or six experts, we have a total of six experts. So when you made your ruling on five, I just looked at the list and said we're going to have to drop
00:50 somebody at this stage and sucked it up and said okay, big deal.

THE COURT: Make it six for each side.

MR. SLATER: Yeah.

THE COURT: Six for each side. There's not going to
00:50 be that much more work.

MR. SLATER: We'll go either way.

THE COURT: Six on each side. So you don't need to send a letter. You have all six.

MS. SHARKO: My other question is if we could have
00:50 the week that we're going to have the Daubert Hearing because

I think we will at least request live testimony so we can make sure that the people we pick are available that week?

THE COURT: You know they don't have to be here to testify live.

00:50 MS. SHARKO: I understand that.

THE COURT: Okay. Well let's work then from you're serving those reports next week. Then depositions you have I assume -- well, I shouldn't assume because plaintiffs haven't seen the defense experts, but assuming the plaintiff wants to

00:51 depose all six.

MR. GOLOMB: Right.

THE COURT: You have 12 depositions to take. How long is this going to take you in February?

MS. SHARKO: We took Judge Schneider at his word.

00:51 That it had to be done in -- by February 28th or we would all die and every one of the experts --

THE COURT: I'm suggesting you don't need that long because you only have 12 now.

MS. SHARKO: No. But, but every one of the experts

00:51 has dates that have been offered. So we have dates set for all --

THE COURT: Okay.

MS. SHARKO: -- of our 40 or how ever many and we have dates from the plaintiffs for all of theirs. So to avoid

00:51 chaos, my request is that we keep the dates that we have.

MR. SLATER: I would say I know that one -- I, I think that it makes sense for us to use the month of February to do the 12 depositions, right. And we just now that with this ruling I assume the defense is going to come back to us
00:51 and tell us which are in and then we can -- we'll know -- then we'll get those dates. We'll know which ones are really in and we'll say yes or no real quick.

THE COURT: When they serve their reports, they're going to tell you the six they're going to rely on.

00:52 MR. SLATER: Whenever they tell us that, we'll say, you know if there's a real -- we're going to try to obviously work with the dates. If there's a major issue, we'll tell them. I doubt it will happen. I mean, I understand the Court's view on that. So -- but it obviously greatly
00:52 simplifies the amount of work we have to do on the briefing because now it's 12 experts instead of across the board. So -- but I don't know how Miss Sharko feels about it. I'm fine with doing the depositions in February and serving the briefs the end of March. If we want to pull the date back a little
00:52 bit because it's only 12 experts, that's fine, too. I, I -- if Your Honor thinks that we don't need as much time, we could probably start writing the briefs now anyway. There's not a lot of secrets to what the standard is. It's going to be dep quotes out of a deposition.

00:52 THE COURT: We all know what the law is.

MR. SLATER: Yup.

THE COURT: We've all been down this road a few times. Well, Miss Sharko, then when are you going to have your motions filed? Do you need as much time as we had previously or can you do it faster?

00:52

MS. SHARKO: We need as much time as we had previously because we've been relying on that date, number one. Number two, we're doing it jointly with New Jersey.

THE COURT: Okay.

00:53

MS. SHARKO: And some of the depositions in New Jersey are staggered. So we're doing two sets of briefs and we would ask March 31 for the briefs and then we propose a schedule back from there, March 31 for motions, April 21, opposition, May 1, reply and the week of May 15 for hearings.

00:53

That's our proposal.

THE COURT: Okay.

MAGISTRATE JUDGE SCHNEIDER: What's the reply date?

THE COURT: May 1st.

MS. SHARKO: May 1st.

00:53

THE COURT: And that's fine. I assume that's okay with the plaintiffs, those dates?

MR. SLATER: I think now that we know what we know what the motions are really and I think those dates work fine. Just to clarify one fine point. The general experts are the same for both litigations. So there's not going to be any

00:53

depositions beyond February because there's no --

THE COURT: Not as to general liability.

MR. SLATER: Right. The general ex -- yeah, the general experts will be deposed in February. There's no more depositions in March.

MS. SHARKO: You're right.

MR. SLATER: Right?

MS. SHARKO: Yeah, you want that on the record. I said you were right.

MR. SLATER: Yeah, that's okay.

THE COURT: Hallelujah. We made progress.

MR. SLATER: You know, Judge, I play chess. I've been moving the chess board for two years to get to that.

THE COURT: Why don't you play baseball.

MR. GOLOMB: That was me.

MR. SLATER: Look at that. Yes, so that's -- so the schedule on that works fine.

THE COURT: Okay.

MR. SLATER: I don't know about the hearing date. That's up to the Court. We can work on that, but the schedule for briefing is fine. Sorry, Carl.

THE COURT: Okay. All right. We have 46 briefs to read and in advance of these. All right. Well, we can do it in the middle of that.

MR. SLATER: Do you want to impose page limits on the

size and the opposition?

MS. SHARKO: I think the Rule fits.

THE COURT: Yeah.

MR. SLATER: Okay, just trying to make it simpler.

00:55 THE COURT: You don't have to repeat the boiler plate legal argument to each and every one of these motions. Okay? These are fact specific. I mean clearly and I think I've hinted at this here and elsewhere, in this kind of a case it seems to me that the qualifications are not going to be an 00:55 issue. It seems to me the fit is not going to be the issue, the methodology is going to be the issue. So let's focus on that. What did they do. How did they get to this opinion. What did they rely on and why is that good science. Do it that way. That's really what I'm interested. So you don't 00:55 have to spend a lot of time in your briefs talking about general law and all the Third Circuit principles and all, except that it applies in that specific fact. Okay?

MS. SHARKO: Okay.

MAGISTRATE JUDGE SCHNEIDER: Miss Sharko, can you 00:56 send me a copy of your six reports to Judge Kugler and myself when you send them to the plaintiff?

MS. SHARKO: Absolutely. We're going to serve all the reports and designate the six? You want all of them with a sticky on our list which ones we're going to use?

00:56 THE COURT: I just want the six for now. The six

that we're going to be dealing with.

MS. SHARKO: Okay, will do.

THE COURT: Okay, we'll start those hearings on May 15th. And I am permitting to you whatever time it takes 00:56 to put on whatever evidence you want, but, you know, don't waste time.

MR. SLATER: I would assume, your Honor, that in advance of those hearings we'll probably be speaking to Your Honor and Judge Schneider about when we get closer about what 00:56 we really think we need to do anyway. Would that be a fair assumption?

THE COURT: Yeah.

MR. SLATER: Okay.

THE COURT: We'll be meeting before then, that's for 00:57 sure. Okay.

MS. SHARKO: In terms of the meetings, I spoke to Mr. Slater before this hearing and our request is that we move the February hearing into March because we're going to be focusing on February depositions albeit fewer than we thought we had.

00:57 THE COURT: Sure.

MS. SHARKO: Okay.

THE COURT: So when in March do you want to do this?

MS. SHARKO: Any day of the week of March 5th is fine for us.

00:57 THE COURT: Well, you want to keep it on a Wednesday

the eighth, the afternoon of the eighth of March? You want to do that?

MR. SLATER: Sure.

MS. SHARKO: Okay. Thank you.

00:57 THE COURT: All right, March 8th at 2:00 p.m.?

MS. SHARKO: Okay.

THE COURT: And obviously if you need a conference before that, we can get you on the phone. All right.

Any other issues we want to talk about today?

00:58 MR. SLATER: I think we've covered everything, Your Honor.

(Brief pause)

THE COURT: All right. Thank you, everybody.

MR. GOLOMB: Thank you, Your Honor.

00:58 MS. SHARKO: Thank you.

THE COURT: Spring training is right around the corner.

(The matter was then concluded)

Exhibit C

Date Prepared: January 31, 2017

Name: Jerrold Ross Turner

Office Address: Thorn Bldg, Room 1428, 20 Shattuck St., Boston, MA 02115

Home Address: 255 Massachusetts Ave, #202, Boston, MA 02115

Work Phone: 617 525 8165 (direct); 847 858 6297 (cell)

Work Email: jrturner@partners.org; jrturner@bwh.harvard.edu

Place of Birth: Chicago, IL

Education

1984	AB magna cum laude	Biology (John P. Atkinson)	Washington University, St. Louis
1984	AM	Biology (John P. Atkinson)	Washington University, St. Louis
1990	PhD	Pathology (Alan M. Tartakoff)	Case Western Reserve University
1991	MD	Medicine	Case Western Reserve University

Postdoctoral Training

07/91-06/93	Resident	Anatomic Pathology	Brigham and Women's Hospital
07/93-09/93	Fellow	Surgical Pathology (Joseph M. Corson)	Brigham and Women's Hospital
10/93-12/93	Chief Resident	Anatomic Pathology (Ramzi S. Cotran)	Brigham and Women's Hospital
01/94-06/95	Clinical Fellow	Gastrointestinal Pathology (James L. Madara and Robert D. Odze)	Brigham and Women's Hospital
01/94-06/95	Postdoctoral Fellow	Gastrointestinal Pathology (James L. Madara)	Brigham and Women's Hospital, Harvard Medical School
01/14-03/14	Visiting Scientist	Laboratory of Host Defense (Warren Strober)	National Institute of Allergy and Infectious Disease (NIAID)

Faculty Academic Appointments

07/95-06/96	Instructor	Pathology	Harvard Medical School
07/96-06/01	Assistant Professor	Pathology	Wayne State University School of Medicine (with voting privileges)
07/01-08/01	Associate Professor	Pathology	Wayne State University School of Medicine (with voting privileges)
09/01-07/03	Assistant Professor	Pathology	The University of Chicago (with voting privileges)
08/03-06/07	Associate Professor (tenured)	Pathology	The University of Chicago (with voting privileges)
07/07-01/16	Professor (tenured)	Pathology	The University of Chicago (with voting privileges)
07/11-01/16	Professor	Medicine (GI)	The University of Chicago (with voting privileges)

07/12-01/16	Sara and Harold Lincoln Thompson Professor		The University of Chicago
02/16-	Appointment Pending	Pathology and Medicine	Harvard Medical School

Appointments at Hospitals/Affiliated Institutions

07/94-06/96	Consulting Pathologist	Pathology	West Roxbury Veteran's Administration Hospital
07/95-06/96	Associate Pathologist	Pathology	Brigham and Women's Hospital
07/96-08/01	Pathologist	Pathology	Detroit Medical Center and Harper Hospital
09/01-01/16	Pathologist	Pathology	The University of Chicago Medical Center
02/16-	Senior Pathologist	Pathology	Brigham and Women's Hospital

Other Professional Positions

1982-84	Undergraduate Research Fellow	Howard Hughes Medical Institute
2002	Scientific Advisory and New Product Development Committee	ICN Biomedicals, Inc
2004-06	Consultant	NPS Pharmaceuticals
2004-10	Consultant	Alba Therapeutics

Major Administrative Leadership Positions

Local

1983	Secretary-Treasurer	Kappa Sigma Fraternity, Beta Sigma Chapter, Washington University, St. Louis
1983	Secretary-Treasurer	Interfraternity Council, Washington University, St. Louis
1984-86	Seminar Series Organizer	Case Western Reserve University, Medical Scientist Training Program
1994	Senior Resident on the Surgical Service	Department of Pathology, Brigham and Women's Hospital
1994	Chief Resident	Department of Pathology, Brigham and Women's Hospital
1994	Clerkship Director (as Chief Resident)	Department of Pathology, Brigham and Women's Hospital
1994-96	Director	Laboratory Sessions, Pathophysiology of Human Disease, Harvard School of Public Health
1995-96	Postdoctoral Fellow Seminar Series Organizer	Harvard Digestive Disease Center

1997-01	Course Director	Gastrointestinal and Liver Pathophysiology (MSII), Wayne State University School of Medicine
2001-04	Director	Cell Biology/Cell Structure Subcore, Digestive Disease Research Core Center (NIH P30), The University of Chicago
2002-03	Organizer	Seminar Series, Committee on Cell Physiology, The University of Chicago
2003-07	Associate Director	Residency Training Program, Department of Pathology, The University of Chicago
2003-07	Course Director	Path315/MOLM 315. Tubes (Gastrointestinal, Respiratory, and Vascular), Graduate Program in Molecular Pathogenesis and Molecular Medicine, The University of Chicago
2004-16	Director	Tissue and Cell Analysis Core, Digestive Disease Research Core Center (NIH P30), The University of Chicago
2004-16	Associate Director	Digestive Disease Research Core Center (NIH P30), The University of Chicago
2007-16	Associate Chair	Department of Pathology, The University of Chicago
2007-9	Course Director	Introduction to Basic & Translational Science Research. Medical Scientist Training Program, The University of Chicago
2011-16	Head	Section of Epithelial Physiology, Institute for Integrative Physiology, The University of Chicago

Regional

2002-06	Founding Chair	Chicago GI Epithelium and Mucosa (GEM) Club

National and International

2005	Organizer (with M. H. Montrose and V. Yang)	Gastrointestinal Tract XI, FASEB Summer Research Conference

2010	Organizer (with J. Nataro)	Bill and Melinda Gates Foundation Symposium on Gut Integrity

Committee Service

Local

1987-89	Seminar Committee	Department of Pathology, Case Western Reserve University, Detroit, MO
1996-01	Research Committee	Department of Pathology, Wayne State University School of Medicine, Detroit, MO
1998-99	Master's Thesis Committee. Christine Perry, laboratory of Dr. Leon Carlock, Ph.D.	Center for Molecular Medicine and Genetics, Wayne State University School of Medicine, Detroit, MO
1999	Space Utilization Review Committee	Department of Pathology, Wayne State University School of Medicine, Detroit, MO
1999-02	Doctoral Thesis Committee. Joseph Krueger, laboratory of Dr. Kaladhar Reddy, Ph.D.	Graduate Program in Cancer Biology, Wayne State University School of Medicine, Detroit, MO
2001-02	Faculty Search Committee (mucosal immunology candidate)	Graduate Program in Molecular Pathogenesis and Molecular Medicine, The University of Chicago, Chicago, IL
2001-04	Doctoral Thesis Committee. Patrick Cullinan, laboratory of Dr. Janis Burkhardt, Ph.D.	Graduate Program in Molecular Pathogenesis and Molecular Medicine, The University of Chicago, Chicago, IL
2002-16	Executive Committee	Committee on Molecular Medicine, The University of Chicago, Chicago, IL
2002-16	Graduate Student Advisory Committee	Graduate Program in Molecular Pathogenesis and Molecular Medicine, The University of Chicago, Chicago, IL
2002-16	Integrated Light Microscopy Core Faculty Advisory Committee	Biological Sciences Division, The University of Chicago, Chicago, IL
2002-09	2007- Medical Scientist Training Program Admissions Committee	Chair Biological Sciences Division, The University of Chicago, Chicago, IL
2002-04	Doctoral Thesis Committee. Alec Vaezi, laboratory of Dr. Elaine Fuchs, Ph.D.	Ben May Cancer Institute, The University of Chicago, Chicago, IL

2002-05	Doctoral Thesis Committee. Michael Puljung, laboratory of Drs. D. Hanck, Ph.D. and E. Beyer, M.D.	Graduate Program in Cell Physiology, The University of Chicago, Chicago, IL
2002-13	Cardiovascular Pathology Training Grant (T32) Internal Advisory Committee	Department of Pathology, The University of Chicago, Chicago, IL
2002-05	Doctoral Thesis Committee. Christine Case, laboratory of Dr. Janis Burkhardt, Ph.D.	Graduate Program in Molecular Pathogenesis and Molecular Medicine, The University of Chicago, Chicago, IL
	2002-05	Chair
2003	Intradepartmental Residency Review Committee	Department of Pathology, The University of Chicago, Chicago, IL
2003-16	Robert Priest Award Selection Committee	Department of Pathology, The University of Chicago, Chicago, IL
2003-16	Pathobiology Seminar Series Committee	Department of Pathology, The University of Chicago, Chicago, IL
	2007-11	Chair
	2012-	Chair
2003-04	Faculty Search Committee (Director of Pathology Informatics)	Department of Pathology, The University of Chicago, Chicago, IL
2003-05	Doctoral Thesis Committee. Miao Zhang, laboratory of Dr. Marcus Clark, M.D.	Graduate Program in Immunology, The University of Chicago, Chicago, IL
	2003-05	Chair
2005-06	Faculty Search Committee (Director of Transfusion Medicine)	Department of Pathology, The University of Chicago, Chicago, IL
2005-08	Faculty Search Committee (Bunning Chair in Food Allergy)	Biological Sciences Division, The University of Chicago, Chicago, IL
2005-09	Doctoral Thesis Committee. Chinonye Nnakwe, laboratory of Dr. S. Kron, M.D., Ph.D.	Department of Molecular Genetics and Cell Biology, The University of Chicago, Chicago, IL
2006	Search Committee (Chairman, Department of Surgery)	Biological Sciences Division, The University of Chicago Biological Sciences Division), Chicago, IL
2006-07	Master's Thesis Committee. Monica Gallas, laboratory of Dr. D. Boone, Ph.D.	Graduate Program in Molecular Pathogenesis and Molecular Medicine, The University of Chicago, Chicago, IL

2007-	Appointments and Promotions Committee (Committee of Professors) 2007-	Department of Pathology, The University of Chicago, Chicago, IL Chair
2007-12	Committee on Appointments and Promotions	Biological Sciences Division, The University of Chicago, Chicago, IL
2007-12	Doctoral Thesis Committee. Tera Lavoie, laboratory of Dr. Julian Solway, M.D.) 2007-12	Graduate Program in Molecular Pathogenesis and Molecular Medicine, The University of Chicago, Chicago, IL Chair
2008-16	Robert Wissler Award Selection Committee	Department of Pathology, The University of Chicago, Chicago, IL
2009	Faculty Science Review Committee	Biological Sciences Division, The University of Chicago, Chicago, IL
2009	Symposium Organizing Committee, "Seeing small is believing big" 2009	Chicago Biomedical Research Consortium, Chicago, IL Chair
2009	Core Services Subcommittee. Biological Sciences Division Faculty Science Review Committee 2009	Biological Sciences Division, The University of Chicago, Chicago, IL Chair
2011-15	Doctoral Thesis Committee. Bryan Berube, laboratory of Dr. J. Bubeck-Wardenburg, M.D., Ph.D.	Graduate Program in Microbiology, The University of Chicago, Chicago, IL
2011-16	Senior Fellow	Institute for Integrative Physiology, The University of Chicago, Chicago, IL
2012-	Doctoral Thesis Committee. Charlie Dulberger, laboratory of Dr. E. Adams, Ph.D.	Graduate Program in Biochemistry, The University of Chicago, Chicago, IL
2012-15	Doctoral Thesis Committee. Joanna Wroblewska, laboratory of Dr. Y.-X. Fu, M.D., Ph.D.	Graduate Program in Immunology, The University of Chicago, Chicago, IL (Committee on Immunology)
2013-15	Doctoral Thesis Committee. Vivian Choi, laboratory of Dr. J. Bubeck-Wardenburg, M.D., Ph.D.	Graduate Program in Microbiology, The University of Chicago, Chicago, IL
2016	Ad hoc Professorial Appointment Committee	Harvard Medical School, Boston, MA

Regional

2002-12	Organizing Committee	Chicago Area GI Epithelium and Mucosa (GEM) Research Group
	2002-06	Chair
2002-04	Doctoral Thesis Committee. Ivana Simnovic, laboratory of Dr. Gail Hecht, M.D.	Department of Microbiology, University of Illinois at Chicago, Chicago, IL
2008	Doctoral Thesis Committee. Rebecca Vongsa-Moyer, laboratory of Dr. M. Dwinell, Ph.D.	Department of Microbiology, Medical College of Wisconsin, Milwaukee, WI
2011	Doctoral Thesis Committee. Harsha Rajapakse, laboratory of Dr. L. W. Miller, Ph.D.	Department of Chemistry, University of Illinois at Chicago, Chicago, IL

National and International

2006	Faculty of 1000	Physiology; gastrointestinal physiology section
2008	Organizing Committee	Molecular structure and function of the tight junction: From basic mechanisms to clinical manifestations, Berlin

Professional Societies

1987-90, 2003-	American Society for Cell Biology	
1995-	Rodger C. Haggitt Gastrointestinal Pathology Society	
	1999-01	Training Committee
	2001-04	Executive Committee
	2002-04	Website Editor/Developer
	2003-06	Co-chair, Publications Committee
1995-	American Gastroenterological Association	
	1998-00	Delegate, Intestinal Disorders Section
	2000	Abstract Selection Committee, *Mechanisms of mucosal injury*
	2001	Abstract Selection Committee, *Intestinal epithelial dynamics and barrier function*
	2001	Abstract Selection Committee, *Epithelial/mesenchymal interactions*
	2001	Chair, Abstract Selection Committee, *Mechanisms of mucosal injury, defense, and repair*

	2002	Abstract Selection Committee, *Inflammation and Epithelial Pathobiology; Injury and Repair*
	2002	Abstract Selection Committee, *Microbial Interactions, Probiotics and Intestinal Host Defense*
	2002-07	Editorial Board Member, *Clinical Gastroenterology and Hepatology*
	2003-04	PhD, MD/PhD, DVM Committee
	2003-05	Chair, Abstract Selection Committee, *Inflammation and Epithelial Pathobiology; Injury and Repair*
	2003-06	Chair, Abstract Selection Committee, *Tight Junctions and Regulation of Barrier Function*
	2004-06	Awards Selection Committee
	2004-07	PhDs and Basic Scientists Task Force
	2006	Chair, Abstract Selection Committee, *Cytoskeleton*
	2006	Chair, Abstract Selection Committee, *Matrix and Mesenchyme*
	2006	Chair, Abstract Selection Committee, *Wound Healing and Repair*
	2006-	Fellow
	2006-07	Nominating Committee
	2009-12	Vice-Chair, Intestinal Disorders Section (elected)
	2009-14	Councilor
	2012-14	Chair, Intestinal Disorders Section (elected)
1995-	U.S.-Canadian Academy of Pathology	
1996-	American Physiological Society	
	2006-09	Councilor, Gastrointestinal and Liver Physiology Section (elected)
	2007	Chair, Special Topic Forum *Roles of Intestinal Epithelia and Bacteria in Inflammatory*

		Disease
	2008	Chair, Cross-Society (with American Society for Experimental Pathology) Special Topic Forum *Regulatory Mechanisms in Diseases of Epithelial Transport*
	2009-12	Secretary/Treasurer, Gastrointestinal and Liver Physiology Section (elected)
	2011	APS Strategic Planning Committee
	2012-15	Chair, Gastrointestinal and Liver Physiology Section (elected)
	2012-15	Section Advisory Committee
2000-	American Society for Investigative Pathology	
	2002-	
	2010-13	Meritorious Awards Committee
	2011-14	Publications Committee
2001-	Crohn's and Colitis Foundation of America	
	2003-06	National Professional Education Committee
2005-	American Society for Clinical Investigation	
	2005-	Member (elected)
2007-	American Society for Biochemistry and Molecular Biology	
2012-	Association of American Physicians	
	2012-	Member (elected)

Grant Review Activities

1997-2006	Merit Award Grant Review (GI)	U.S. Veteran's Administration
	1997, 1998, 1999, 2000, 2001, 2002	External Reviewer
	2003-06	Member
2001	NIDDK Special Emphasis Panel, Digestive Diseases Research Core Centers GRB-4	NIH
	2001	Member
2002	Special Emphasis Review Panel, Centers of Biomedical Research Excellence (COBRE)	NIH
	2002	Member

2002-03	Special Emphasis Panel ZRG1ALTX-1 2002, 2002, 2003	NIH Member
2002-18	NIDDK Sub-Committee C 2002, 2003, 2003, 2013 2004-10 2008-09 2014-18	NIH Ad hoc member Permanent member Chair Permanent member
2003-08	2003, 2005, 2006, 2007, 2008	Broad Medical Research Foundation Grant Proposal Reviewer (External)
2004	Special Emphasis Panel ZDK GRB-6 (02) 2004	NIH Member
2004-11	NIH Special Emphasis Panel, ZRG F10 DIG-C 2004, 2004, 2005, 2005, 2006, 2008, 2011	NIH Member
2004-15	Senior Research Award Review Panel 2004, 2005, 2006, 2007, 2008 2009-15	Crohn's and Colitis Foundation of America Grant Proposal Reviewer (External) Permanent member
2005-06	Clinical and Integrative Gastrointestinal Pathobiology Study Section 2005, 2006	NIH Ad hoc member
2005-08	External Reviewer 2005, 2007, 2008	Science Foundation Ireland Grant Proposal Reviewer (External)
2005-09	Special Emphasis Panel. ZRG1 DIG-C 2005, 2005, 2007, 2007, 2009	NIH Member
2007	External Reviewer	Italian Cystic Fibrosis Research Foundation
2010-12	NIDDK Special Emphasis Panel, Nutrition Obesity Research Centers (P30) 2010, 2012	NIH Member
2010-13	Digestive Disease and Nutrition Pre- and Postdoctoral Fellowships 2010, 2011, 2012, 2013	NIH Ad hoc member

2011	Special Emphasis Panel, ZRG1 DKUS-C (03) 2011	NIH Chair
2011	NIAID Special Emphasis Panel Immune Defense Mechanisms at the Mucosa Cooperative Study Group (U01) ZAI1WFD-I (M2) 2011	NIH Ad hoc member
2011-12	Congressionally Directed Medical Research Program (CDMRP), Inflammatory Bowel Disease 2011, 2012	Department of Defense Ad hoc member
2012	RFA RM11-006: Transformative R01 Roadmap Review (R51) ZRG1 BCMB-A (51) R 2012	NIH External reviewer
2012-13	Special Emphasis Panel, NIDDK Committee C Conflicts ZDK1 GRB-2 2012, 2012, 2013	NIH Member
2013	Gastrointestinal Mucosal Pathobiology Study Section 2013	NIH Ad hoc member
2013	Pilot and Feasibility: Clinical Research Studies in Digestive Diseases and Nutrition (R21) ZRG1 DKUS 2013	NIH Member
2013	Mid-Term Site Visit, Principal Investigator award "Molecular Mechanisms of Epithelial Transport" Royal College of Surgeons in Ireland (RCSI) 2013	Science Foundation Ireland Member
2013-15	PREPARE (Peer Review by Experts Promotes Achieving Research Excellence) Pilot Program 2013, 2015	Pennsylvania State University College of Medicine External reviewer
2013	Blanc program 2013	French Research Agency (ANR) External reviewer
2013	Discovery Grant Program 2013	Natural Sciences and Engineering Research Council of Canada (NSERC) External reviewer
2014	Special Emphasis Panel, ZRG1 DKUS A 10 2014	NIH Member

2014	Special Emphasis Panel, Mechanisms integrating organismal, tissue, and cellular aging 1 P01 AG048818-01 2014	NIH Member

Editorial Activities

Ad hoc Reviewer

American Journal of Gastroenterology
American Journal of Pathology
American Journal of Pathology
American Journal of Physiology - Cell Physiology
American Journal of Physiology - Gastrointestinal and Liver Physiology
American Journal of Respiratory Cell and Molecular Biology
Biochemical Pharmacology
BMC Cell Biology
BMC Medicine
Cancer
Cancer Research
Cell
Cell and Molecular Life Sciences
Cell Host and Microbe
Cell Motility and the Cytoskeleton
Clinical Cancer Research
Clinical Gastroenterology and Hepatology
Current Biology
FASEB Journal
Gastroenterology
Gut
Infection and Immunity
Inflammatory Bowel Disease
International Journal of Biochemistry and Cell Biology
International Journal of Cancer
International Journal of Experimental Diabetes Research
Journal of Biological Chemistry
Journal of Cell Biology
Journal of Cell Science
Journal of Cellular Biochemistry
Journal of Clinical Investigation
Journal of Comparative Physiology
Journal of Experimental Medicine
Journal of Immunology
Journal of Inflammatory Bowel Disease
Journal of Pathology
Journal of Physiology
Microvascular Research
Modern Pathology
Molecular and Cellular Biology

Molecular Biology of the Cell
Mucosal Immunology
Nanoscale Biochemical Journal
Nature Immunlogy
New England Journal of Medicine
PLoS Biology
PLoS ONE
Proceedings of the National Academy of Sciences U.S.A.
Science
Science Translational Medicine
Trends in Microbiology
Zoology

Other Editorial Roles

2003-	Editorial Board Member	*American Journal of Physiology - Gastrointestinal and Liver Physiology*
2003-	Editorial Board Member	*American Journal of Pathology* (gap during service as Associate Editor, 2013-14)
2004-	Editorial Board Member	*Laboratory Investigation* (gap during service as Senior/Associate Editor, 2004-06)
2004-06	Associate Editor	*Laboratory Investigation*
2006	Guest Editor, "Clinical Pathology for the Gastroenterologist and Hepatologist"	*Clinical Gastroenterology and Hepatology*
2006-08	Senior Associate Editor	*Laboratory Investigation*
2006-08	Section Editor, "Pathobiology in Focus"	*Laboratory Investigation*
2007-	Editorial Board Member	*Gastroenterology* (gap during service as Associate Editor, 2011-14)
2008-13	Editorial Board Member	*Journal of Biological Chemistry*
2011-14	Associate Editor	*Gastroenterology* (selected via competitive process; resigned due to new position as Editor of *Cellular and Molecular Gastroenterology and Hepatology*)
2013-14	Associate Editor	*American Journal of Pathology* (resigned due to new position as Editor of *Cellular and Molecular Gastroenterology and Hepatology*)
2014-19	Editor-in-Chief (Founding)	*Cellular and Molecular Gastroenterology and*

		Hepatology (selected via competitive process)	
2015-20	Editorial Board Member	*Journal of Biological Chemistry*	

Honors and Prizes

1983	National Scholarship and Leadership Award	The Kappa Sigma Fraternity	
1983	Student Research Fellowship	Howard Hughes Medical Institute	
1984-91	Trainee, Medical Scientist Training Program	National Institutes of Health	
1990	First Prize, Irwin H. Lepow Student Research Day	Case Western Reserve University	
1991	The Martin Wahl Memorial Fund Award	Case Western Reserve University	*"for independence and excellence in research and clinical skills"*
1996-2001	Mentored Clinical Scientist Career Development Award (K08)	National Institutes of Health	
1999	Faculty Research Excellence Award	Wayne State University School of Medicine	
1999	Excellence in Pathology Education Award	Wayne State University School of Medicine	elected by the MSII class
2000	College Teaching Award	Wayne State University	
2002	Senior Research Award	Crohn's and Colitis Foundation of America	
2005	Elected Member	American Society for Clinical Investigation	
2006	Amgen Outstanding Investigator Award	American Society for Investigative Pathology	
2006-	Fellow	American Gastroenterological Association	
2006-	Member, Faculty of 1000	Physiology; gastrointestinal physiology section	

2011	Takeda Distinguished Research Award	Gastrointestinal and Liver Physiology Section, American Physiological Society	
2011	Intestinal Disease Research Unit (IDRU) Lectureship in Inflammatory Bowel Disease	Gastrointestinal Research Unit, University of Calgary	*Stopping the flood: The gut barrier and its role in inflammatory bowel disease*
2012-	Sara and Harold Lincoln Thompson Professor	The University of Chicago	
2012-	Elected Member	Association of American Physicians.	
2012	Daljit and Elaine Sarkaria Lecturer	Department of Pathology, University of California, Los Angeles	
2015	Hans Ussing Lecturer	Epithelial Transport Group, American Physiological Society	*"to recognize scientists who have made fundamental contributions to our understanding of epithelia transport and diseases of epithelial transport"*
2015	Research Mentor Award	Intestinal Disorders Section, American Gastroenterological Association	

Report of Funded and Unfunded Projects

Funding Information

Past

1/1/95 – 6/30/96

Tight junction regulation in intestinal epithelium
F32 DK09180
$49,400
The goal of this proposal was to study molecular mechanisms of tight junction regulation. The three complementary aims were to 1) stably transfect human intestinal epithelial cell lines with the human intestinal Na^+-glucose cotransporter SGLTl in order to develop a model of physiologic tight junction regulation; 2) characterize the physiologic response of SGLT1 transfectants to apical glucose; and 3) examine the role of rab13 in the functional regulation of tight junctions using a panel of developmentally, physiologically, and pathophysiologically relevant stimuli. This award was terminated when the K08 DK02463 began.

7/1/96 – 8/30/01
Mechanisms of intestinal tight junction regulation
K08 DK02463
$519,500
The aims of this proposal were to 1) characterize proposed signal transduction pathway leading from Na^+-nutrient cotransport to altered TJ permeability using an enterocyte-like cell line (Caco-2) stably transfected with the intestinal Na^+-glucose cotransporter SGLT1; and 2) evaluate structural and biochemical changes in TJ composition that follow activation of Na^+-glucose cotransport.

7/1/99 – 6/30/01
Tight junction regulation by myosin light chain kinase
R03 DK56121
$150,000
The aims of this proposal were to define and characterize the molecular mechanisms by which MLC phosphorylation regulates TJ permeability.

1/1/00 – 12/31/01
Cytoskeletal regulation of intestinal wound healing
Children's Research Center of Michigan
$100,000
The aim of this pilot and feasibility award were to define the role(s) of myosin light chain kinase in epithelial wound healing.

9/1/01– 6/30/04
Biology and pathobiology of GI epithelial cells
P30 DK42086
$3,302,003; Director of Cell Biology/Cell Structure
This award supported The University of Chicago Digestive Disease Research Core Center.

9/1/01 – 6/30/06
Physiological regulation of intestinal permeability
R01 DK61931
$1,517,912
The aims of this proposal were to 1) determine the signaling events that link Na^+-glucose cotransport to NHE3 activation; 2) characterize the roles of human intestinal epithelial MLC kinase isoforms in tight junction regulation; and 3) characterize the effects of MLC phosphorylation on perijunctional actomyosin ring organization and tight junction structure and function.

7/1/02 – 6/30/05
Barrier dysfunction in Crohn's disease: Myosin light chain phosphorylation as mediator and therapeutic target
Senior Research Award, Crohn's & Colitis Foundation of America
$345,000
The aims of this proposal were to 1) characterize the biochemical mechanisms by which the myosin light chain kinase inhibitor PIK prevents tight junction permeability increases induced by pathophysiological stimuli; 2) determine effects of PIK on cell migration and tight junction resealing; and 3) generate a membrane permeant MLC kinase inhibitor with stability suitable for in vivo use.

7/1/04 – 11/30/05
Biology and pathobiology of GI epithelial cells.
P30 DK42086
$1,000,001; Associate Center Director; Director of the Tissue and Cell Analysis Core

This award supported The University of Chicago Digestive Disease Research Core Center.

7/1/05 – 6/30/10
Regulation of paracellular permeability by IFNγ and TNFα
R01 DK68271
$1,714,154
The aims of this proposal were to 1) determine the role of IFNγ in enhancing epithelial responsiveness to TNFα and the mechanisms by which IFNγ and TNFα synergize to increase MLC phosphorylation; 2) define the effects of IFNγ and TNFα on tight junction protein dynamics; and 3) characterize the effects of IFNγ and TNFα on the regulation of MLC phosphorylation in vivo and in situ using TNFR1, TNFR2, and MYLK knockout mice.

12/1/05 – 11/30/15
IBD and mucosal inflammation; immunology; and microbiology.
P30 DK42086
$11,553,424; Associate Center Director; Director of the Tissue and Cell Analysis/Molecular and Experimental Pathology Core ($1,557,864).
This award supported The University of Chicago Digestive Disease Research Core Center.

5/1/06 – 4/30/07
Approaches for oral drug delivery
Unity Pharmaceuticals, Inc
$50,000
The goals of this sponsored research agreement were to assess approaches for oral delivery of a novel membrane permeant MLC kinase inhibitor.

7/1/06 – 6/30/07
Physiological Regulation of Intestinal Epithelial Transport and Barrier Function
R01 DK061931-06S1
$17,066
This supplement supported a minority undergraduate student.

7/1/06 – 6/30/11
Physiological regulation of intestinal permeability
R01 DK61931
1,587,326
The aims of this proposal were to 1) define the role of ezrin in acute regulation of protein delivery to the plasma membrane; 2) identify the mechanisms that define the dynamic behavior of proteins at the tight junction; and 3) define the mechanisms and significance of actomyosin-dependent tight junction maintenance and regulation.

1/1/08 – 12/31/09
Multiplexed imaging of transient molecular complex dynamics in vivo
Catalyst Award, Chicago Biomedical Consortium
$200,000; Co-PI (MPI)
The aim of this collaborative proposal was to assess the potential of a time-resolved fluorescence resonance energy transfer approach to analyze tight junction protein interactions in living cells.

1/1/08 – 12/31/10
Epithelial tight junction regulation and immune activation in inflammatory bowel disease
Research Fellowship Award, Crohn's & Colitis Foundation of America
$159,033
This award was to support training of Dr. L. Shen within Dr. Turner's laboratory. It

focused on the contributions of enhanced tight junction permeability to progression of experimental inflammatory bowel disease.

7/1/08 – 6/30/10
Mechanisms of epithelial barrier dysfunction mediated by inflammatory cytokines
F32 DK082134
$108,208; Mentor
This award was to support training of Dr. C. Weber within Dr. Turner's laboratory. It focused on the biophysical characteristics and mechanisms of tight junction regulation by pro-inflammatory cytokines. This fellowship was terminated prematurely to allow Dr. Weber to accept a K08 award.

5/1/09 – 10/30/12, 9/1/13 – 8/31/14
Epithelial myosin light chain kinase trafficking: a therapeutic target in inflammatory bowel disease
IBD-022, Broad Medical Research Foundation
$299,911
The aims of this proposal were to 1) define the contributions of the epithelial long myosin light chain kinase isoform 1 (MLCK1) to inflammaory bowel disease pathogenesis; and 2) characterize a novel approach to interrupt MLCK1 trafficking to the tight junction to prevent MLCK1-dependent barrier dysfunction.

6/1/09 – 5/31/12
Epithelial barrier-dependent mucosal immune regulation: Mechanisms and interventions
W81XWH-09-1-0341, Department of Defense CDMRP
$1,385,840
The aims of this proposal were to 1) determine how innate mucosal immune regulation is altered following epithelial barrier dysfunction; 2) determine how the function of adaptive regulatory cells within the mucosal immune system are modulated by epithelial barrier dysfunction; and 3) determine the roles of innate (DCs) and adaptive (Tregs) immune cells in maintaining in vivo immune homeostasis and preventing disease despite epithelial barrier dysfunction.

7/20/09 – 6/30/11
Physiological regulation of intestinal epithelial transport and barrier function
R01 DK061931-08S1
$56,338
This supplement was awarded under the American Recovery and Reinvestment Act to support summer trainees.

9/30/09 – 9/29/11
Immune cell regulation of intestinal epithelial barrier function during colitis
F32 DK084859
$87,146; Mentor
This award was to support training of Dr. K. Edelblum within Dr. Turner's laboratory. It focused on function of intraepithelial lymphocytes and their interactions with the intestinal epithelium in vivo.

12/03/09 – 6/30/10
Regulation of paracellular permeability by IFNγ and TNFα
DK068271-05S1
$99,959
This supplement was awarded under the American Recovery and Reinvestment Act to support a postdoctoral fellow and new equipment related to the parent grant.

1/1/10 – 12/31/13	Occludin-dependent regulation of epithelial apoptosis in IBD Mentored Career Development Award, Crohn's & Colitis Foundation of America $270,000; Mentor This award was to support continued training of Dr. L. Shen within Dr. Turner's laboratory. It focused on the role of occludin in directing epithelial apoptosis.
7/01/10 – 6/30/11	Physiological Regulation of Intestinal Epithelial Transport and Barrier Function R01 DK061931-10S1 $95,810 This supplement awarded under the American Recovery and Reinvestment Act is for purchase of a new microscope to support studies supported by DK061931, DK068271, DK067887, DK088953, and DK084859.
7/1/10 – 6/30/15	Mechanisms and pathways of trans-tight junction conductance K08 DK088953 $732,780; Mentor This mentored career development award was to support continued training of Dr. C. Weber within Dr. Turner's laboratory. It focused on development of a patch clamp approach to detect single channel tight junction conductance events.
7/16/10 – 7/15/11	Advanced Multi-color Confocal and FRAP-SAC Microscope S10 RR025643 $499,909 This application supported the purchase of a spinning disk confocal microscope with spherical aberration correction for the Biological Sciences Division Integrated Light Microscopy Core.
5/1/11 – 9/01/11	Talactoferrin interactions of with intestinal epithelial cells Agennix, Inc $11,295 The goals of this sponsored research agreement were to assess the direct interactions of recombinant talactoferrin with epithelial cell surfaces.
7/01/11 – 6/30/13	Immune cell regulation of intestinal epithelial barrier function during colitis Research Scholar Award, American Gastrointestinal Association $120,000; Mentor This mentored award was to support continued training of Dr. K. Edelblum within Dr. Turner's laboratory. It focused on function of intraepithelial lymphocytes and their interactions with the intestinal epithelium in vivo. This award was terminated prematurely to allow Dr. Edelblum to accept a K01 career development award.
7/01/11 – 6/30/12	Role of MLCK1 trafficking in epithelial barrier regulation F32 DK091017 $48,398; Mentor This fellowship award was to support training of Dr. E. Bradford within Dr. Turner's laboratory. It focused on trafficking of MLCK1 within intestinal epithelium.

8/21/06 – 7/31/12	Regulation of intestinal transport $597,752; Cell Imaging Core Director This multi-investigator award supports a collaborative research program focused on intestinal transport.
9/1/12 – 8/31/13	HTS assays for inhibitors of protein interactions including MLCK1-FKBP8 R56 DK094954 $54,600; Co-PI (MPI) This multi-PI grant focused on developing novel high throughput assays to detect protein-protein interactions.
7/1/11 – 6/30/14	Mechanisms and consequences of cytokine-induced tight junction barrier regulation R01 DK68271 $1,956,599 The goals of this proposal were to 1) characterize the molecular mechanisms of MLCK activation during 'leak' pathway regulation; 2) define the mechanisms that regulate 'pore' pathway flux; and 3) elucidate the distinct roles of 'pore' and 'leak' pathways in disease.
7/01/14 – 6/30/15	Role of Cronobacter sakazakii in necrotizing enterocolitis Research Scholar Award, American Gastrointestinal Association $150,000; Mentor This mentored award was to further the career development of Dr. C. Hunter. It focused on mechanisms of intestinal epithelial dysfunction in necrotizing enterocolitis.
7/1/11 – 6/30/15	Molecular mechanisms of intestinal epithelial tight junction regulation R01 DK61931 $2,334,960 The goals of this proposal were to define the mechanisms by which signal transduction pathways that activate membrane traffic and modify protein interactions are involved in regulation of intestinal transport and barrier function
1/1/13 – 12/31/15	Claudin-2 in barrier loss and IBD: Pathogenic impact and therapeutic potential Senior Research Award, Crohn's & Colitis Foundation of America $343,953 The major goal of this proposal was to determine the contribution of increased claudin-2 expression to IBD progression and to assess the therapeutic potential of blocking claudin-2 pore permeability.
6/1/13 – 3/15/15	Identification of novel biomarkers for environmental enteropathy in children using an evidence based approach OPP1066200, Bill & Melinda Gates Foundation $56,810 The goal of this award is to develop biomarkers for diagnosis and treatment of environmental enteropathy. The primary grant was awarded to Dr. Syed Asad Ali, Assistant Professor of Paediatrics and Child Health, Aga Khan University, Karachi, Pakistan.
2/1/14–	Environmental enteropathy in Zambia: biomarkers defined by pathogenesis

1/31/15	OPP1066118, Bill & Melinda Gates Foundation $76,192 The goal of this award is to develop biomarkers for diagnosis and treatment of environmental enteropathy. The primary grant was awarded to Dr. Paul Kelly, Reader in Tropical Gastroenterology, Centre for Digestive Diseases, Blizard Institute, Queen Mary & Westfield College, Barts and the London School of Medicine and Dentistry, United Kingdom, and University of Zambia, Lusaka, Zambia.
12/1/10 – 11/30/15	Biology and pathobiology of GI epithelial cells. P30 DK42086 Associate Center Director; Director of the Tissue and Cell Analysis Core This award supported The University of Chicago Digestive Disease Research Core Center.
9/1/11 – 10/28/14	ZO-1 domain interactions in tight junction structure and barrier function F32 DK094550 $147,341; Mentor This fellowship award was to support training of Dr. M. Buschmann within Dr. Turner's laboratory. It focused on the function of ZO-1 in tight junction regulation.
Current	
2/15/12 – 1/31/17	γδ IEL migration and epithelial interactions in intestinal disease K01 DK093627 $735,210; Mentor This mentored career development award was to support continued training of Dr. K. Edeblum within Dr. Turner's laboratory. It focused on roles of γδIEL migration in innate immune function. Dr. Edelblum accepted a position as Assistant Professor of Pathology at Rutgers University effective 7/1/15. Dr. Turner remains involved as the mentor.
7/1/12 – 6/31/17	Contribution of occludin to intestinal homeostasis and disease K01 DK092381 $750,760; Mentor This mentored career development award is to support continued training of Dr. L. Shen within Dr. Turner's laboratory. It focuses on the role of occludin in directing epithelial apoptosis.
6/1/14 – 5/31/17	Cytoskeletal mechanisms of epithelial morphogenesis F30 DK103511 $75,718; Mentor This fellowship award was to support M.D.-Ph.D. student Matthew Odenwald during Ph.D. training within Dr. Turner's laboratory and for the final two years of medical school. It focused on the function of ZO-1 in regulating the cytoskeleton during epithelial morphogenesis.
7/1/14 – 6/30/19	The myosin light chain kinase-phosphatase axis in GI homeostasis and disease R01 DK68271 $2,350,260 The objectives of this application are to 1) characterize the molecular mechanisms of MLCK1 interactions with the molecular chaperone FKBP8; 2) define the mechanisms by

which TNF induces MLCK1 trafficking; and 3) to define the roles of MYPT1 in epithelial homeostasis.

7/1/14 – 6/30/19
Translational approaches to develop drug therapy for diarrhea
R24 DK099803
$8,894,205
The objectives of this multi-institutional program are to 1) catalog and identify potential molecular targets for anti-diarrheal therapy in human disease; 2) to define the mechanisms and contributions of functional changes in small intestinal ion absorption and secretion in diarrhea; and 3) to test the efficacy of novel anti-secretory and pro-absorptive therapeutics in disease-relevant diarrhea models. Dr. Turner is responsible for the first aim (1,975,000).

7/1/15 – 6/30/20
ZO-1-mediated protein interactions as regulators and integrators of diverse epithelial functions in health and disease.
R01 DK61931
$3,062,060
The objectives of this application are to define the mechanisms by which ZO-1 integrates diverse aspects of epithelial function, including morphogenesis, barrier function, and wound repair.

7/1/15 – 6/31/20
Defining bacterial virulence, cAMP and PKA in necrotizing enterocolitis
K08 DK106450
$727,434; Mentor
This mentored career development award is to support continued training of Dr. C. Hunter. It focuses on mechanisms of *Cronobacter sakazakii* signaling to induce intestinal epithelial dysfunction in necrotizing enterocolitis.

12/1/15 – 11/30/20
IBD and mucosal inflammation, immunology, and microbiology of the GI tract.
P30 DK42086
$5,850,000; Associate Center Director; Director of the Tissue and Cell Analysis Core ($640,350).
This award supports The University of Chicago Digestive Disease Research Core Center.

Report of Local Teaching and Training

Teaching of Students in Courses

1990	Physical Diagnosis Year 2 Medical Students	Case Western Reserve University Laboratory Instructor, 20 hours of direct contact
1992	Introduction to Pathology Year 2 Medical Students	Harvard Medical School Laboratory Instructor, 3 hours of direct contact
1993	Pathology Clerkship Year 3/4 Medical Students	Brigham and Women's Hospital Clerkship Director (as Chief Resident) , 30 hours of direct contact
1993	Human Pathology	Harvard-MIT program in Health Sciences

	Year 2 Medical Students	and Technology Laboratory Instructor, 3 hours of direct contact
1993	Infectious Disease Year 2 Medical Students	Harvard Medical School Laboratory Instructor, 3 hours of direct contact
1994-96	Gastrointestinal Pathophysiology. Year 2 Medical Students	Harvard Medical School Laboratory Instructor, 3 hours of direct contact
1995-96	Gastrointestinal Pathology and Liver, Biliary, and Pancreatic Pathology. Pathophysiology of Human Disease MPH and PhD students	Harvard School of Public Health Lecturer and Laboratory Director, 20 hours of direct contact
1996-2000	NBME review course Year 2 Medical Students	Wayne State University School of Medicine Lecturer, 4 hours of direct contact
1996-2000	Systemic Pathophysiology. Pathology 650 Year 1/2 Medical Students	Wayne State University School of Medicine Lecturer, 6 hours of direct contact
1997-2001	Gastrointestinal Pathophysiology Year 2 Medical Students	Wayne State University School of Medicine Course Director and Lecturer, 33 hours of direct classroom contact and 20 hours with individual students and small groups
1998-99	Cell Biology. IBS 7020 Year 1/2 PhD Students	Wayne State University School of Medicine Discussion group leader, 4 hours of direct contact
1998-2000	Biomedical gastrointestinal system biology and nutrition. IBS 7080 Year 1/2 PhD Students	Wayne State University School of Medicine Lecturer, 8 hours of direct contact
2000-01	Approach to Gross Examination Pathology Assistant B.A. Students	Wayne State University School of Mortuary Science Lecturer, 8 hours of direct contact
2001-04	Medical Cell Biology: Cell Junctions Year 1 PhD Students	The University of Chicago Lecturer, 6 hours of direct contact
2002-07	Gastrointestinal Histology Year 1 MD-PhD Students	The University of Chicago Lecturer, 6 hours of direct contact
2002-10	Clinical Pathophysiology and Therapeutics: Pathology of Small Intestinal Causes of Malabsorption	The University of Chicago

	Year 2 Medical Students	Lecturer, 3 hours of direct contact
2002-13	Clinical Pathophysiology and Therapeutics: Pathology of Inflammatory Bowel Disease Year 2 Medical Students	The University of Chicago Lecturer, 3 hours of direct contact
2003-07	Tubes. Path315/MOLM 315 Year 1/2 PhD Students	The University of Chicago Co-course director and Lecturer, 20 hours of direct contact
2005	Cell Injury, Repair and Death. MPMM 57500-01: Tissue response to injury and repair induction Year 1/2 PhD Students	The University of Chicago Lecturer, 3 hours of direct contact
2005	Molecular Nutrition. MOMN 366: Mechanisms of Nutrient Absorption Year 1/2 PhD Students	The University of Chicago Lecturer, 3 hours of direct contact
2005-07	Cell Biology 302. Cytoskeleton and Cell Junctions Year 1 Medical Students	The University of Chicago Lecturer, 4 hours of direct contact
2007-09	Introduction to Basic & Translational Science Research Year 1 MD-PhD Students	The University of Chicago Course director and Lecturer, 16 hours of direct contact
2008-09	Molecular and Cell Biology. SURG 30302-01: Cytoskeleton and cell junctions Year 1 MD-PhD Students	The University of Chicago Lecturer, 2 hours of direct contact
2010-12	Cells, Molecules, and Genes MS1: Epithelial polarity and barrier function Year 1Medical Students	The University of Chicago Lecturer, 2 hours of direct contact
2011-15	Mucosal Immunology. IMMU 35500: The epithelial barrier. Year 1/2 PhD Students	The University of Chicago Lecturer, 3 hours of direct contact

Formal Teaching of Residents, Clinical Fellows and Research Fellows (post-docs)

1996-2001	Surgical Pathology Unknown Conferences Pathology Residents	Wayne State University School of Medicine Lecturer, 3 hours of direct contact
2001-08	Surgical Pathology Unknown Conferences Pathology Residents	The University of Chicago Lecturer, 1 hour of direct contact
2001-07	Molecular Pathology Conference Pathology Residents	The University of Chicago Teacher, 2-3 hours of direct contact before each 1 hour lecture (lectures by residents)

Clinical Supervisory and Training Responsibilities

1996-2001	Surgical and Autopsy Pathology Preceptor, Wayne State University School of Medicine	120 days per year (~1,000 cases)
2001-	Gastrointestinal Surgical Pathology Preceptor	35 days per year (~1,000 cases)

Formally Supervised Trainees

1996-97 Brian K. Rill, M.D./Attending Orthopedic Surgeon, Henry Ford Health System
Research Assistant who assisted with initial studies of myosin light chain kinase and physiological tight junction regulation. Was co-author on one paper published in *Amer J Physiol – Cell Physiol.*

1996-97 Rafael E. Jimenez, M.D./Assistant Professor of Pathology, Mayo Clinic
Pathology resident who studied the relationship between dialysis and gastrointestinal amyloidosis. Published one first author paper in *Amer J Surgical Path.*

1997-98 Gail Bentley, M.D./Assistant Professor of Pathology, Wayne State University School of Medicine
Pathology resident who studied the role of race as a risk factor in HCV hepatitis. Won the Hans Popper Hepatopathology Society Resident Research Award at the 1998 US-Canadian Academy of Pathology Meeting.

1999-2001 Yevgeny Zolotarevsky, M.D. /Attending Gastroenterologist, St. Joseph Mercy Hospital, Ann Arbor, MI
Undergraduate Student who studied the role of myosin light chain phosphorylation in TNF-induced barrier loss. Published one first author paper in *Gastroenterology.* After graduation from Wayne State University, attended medical school at Wayne State University and internal medicine residency and gastroenterology fellowship at the University of Michigan.

1999-2001 Hui-Ren Zhao, M.D./Senior Research Associate, Wayne State University School of Medicine
Postdoctoral fellow who studied mechanisms of NHE3 activation during Na^+-nutrient cotransport. Published one first author paper in *PNAS.*

2000 Jessica J. Berglund/Current Position Unknown
Summer Undergraduate Research Experience (SURE) Fellow who studied the Role of myosin light chain phosphorylation in human small intestine during Na^+-nutrient cotransport. After graduating from Hope College matriculated at University of Michigan Medical School. Published one first author paper in *Amer J Physiol – GI Physiol.*

2000 Shari Rosen, Pharm.D./Infectious Diseases Clinical Pharmacist, Capital Health, Medical Center, NJ
High School Xtudent who studied myosin light chain kinase splice variants. Published one first author paper in *J Biol Chem.* received undergraduate degree and Pharm.D. from the University of Michigan.

2002-04 John Russo, M.D /Assistant Professor of Clinical Pediatrics, The Ohio State University School of Medicine
Pediatric Gastroenterology Fellow who studied mechanisms of wound closure and published one first author paper in *Gastroenterology* and a book chapter.

2002-06 Daniel Clayburgh, M.D.-Ph.D./Assistant Professor of Otolaryngology, Oregon Health Sciences University
Medical Scientist Training Program (MSTP) Student who studied myosin light chain kinase and NHE3 in diarrheal disease and published two first author papers *J Clinical Invest*, one first author paper in *J Biol Chem* as well as thirteen additional first and middle articles and book chapters. Was awarded multiple abstract prizes.

2002-16 Le Shen, M.D., Ph.D./Research Associate (Assistant Professor), Department of Pathology, The University of Chicago
Ph.D. Student, Postdoctoral Fellow, and Faculty member who studied tight junction protein dynamic behavior and occludin function, in vitro and in vivo. Published three first author papers (*J Cell Biol, Mol Biol Cell, J Cell Sci*), a first author review in *Annual Review of Physiology*, and 34 other first author and collaborative works. Won The University of Chicago Robert Priest Award and was funded by Crohn's Colitis Foundation of America Research Fellowship, Crohn's Colitis Foundation of America Career Development Award, and NIH K01.

2003-04 Fengjun Wang, M.D., Ph.D./Professor of Burn Surgery, State Key Laboratory of Trauma, Burns & Combined Injury, Institute of Burn Research, Southwest Hospital, Third Military Medical University, Chongqing, China
Postdoctoral fellow who studied the role of myosin light chain kinase and TNF receptors in cytokine-induced barrier loss and published one first author paper each in *Gastroenterology* and *Amer J Pathol*, a co-first author paper in *Gastroenterology*, and two other first and middle papers.

2003-05 Edwina Witkowski (Schmitz), D.V.M./Attending Veterinarian (private practice), Nebraska
Research Assistant who studied Rho kinase regulation of barrier function.

2004-05 Stephanie Blair (Deal), M.D./Instructor in Pediatrics (Neonatology), Baylor College of Medicine
Undergraduate Student and summer Howard Hughes Medical Institute Research Fellow who studied myosin light chain kinase expression and activity in IBD and published one first author paper in *Lab Invest*. After graduation from The University of Chicago, attended medical school at Baylor College of Medicine and completed pediatrics residency at Lurie Children's Hospital/Northwestern University.

2005-06 Zhihong Hu, M.D., Ph.D./Pathology Resident Loyola University (Chicago)
Postdoctoral fellow who studied mechanisms of NHE3 activation during Na^+-nutrient cotransport and published one first author paper in *J Biol Chem* as well as a book chapter.

2005-07 Brad T. Schwarz, D.O./Anesthesiology Resident, University Hospitals, Cleveland, OH
Research Assistant who studied the role of myosin light chain kinase and TNF receptors in cytokine-induced barrier loss and published one first author paper each in

Gastroenterology, one a co-first author paper in *Gastroenterology*, and was co-author on two additional papers (*Amer J Pathol* and *J Cell Biol*). Subsequently earned medical degree and joined anesthesiology residency at University Hospitals, Cleveland/Case Western Reserve University.

2005-08 Liping Su, M.D./Associate Professor, Shanghai Institute of Digestive Surgery, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China
Postdoctoral fellow who studied myosin light chain kinase in experimental IBD and published two first author papers in *Gastroenterology* as well as a first author commentary in *Gastroenterology* and five co-authored articles.

2005-10 Dan Yu, M.D., Ph.D./Postdoctoral Fellow, University of California San Francisco
Ph.D. Student who studied myosin light chain kinase, ZO-1 trafficking, and barrier regulation, published one first author article in *PNAS,* a first author review, and six other collaborative works. Won American Gastroenterological Association Research Award.

2005-10 Amanda Marchiando, Ph.D., M.P.H./Postdoctoral Fellow, New York University
Ph.D. Student who performed in vivo analysis of intestinal epithelial barrier maintenance, regulation, and repair and published first author papers in *J Cell Biol* and *Gastroenterology*, a first author review in *Annual Review of Pathology*, and seven other collaborative works. Won American Gastroenterological Association and American Physiological Society Research Awards.

2005-10 W. Vallen Graham, Ph.D., M.S./Postdoctoral Fellow, Rockefeller University
Ph.D. Student who studied myosin light chain kinase expression and trafficking and published two first author papers, one in *J Biol Chem*, a co-first author paper in *Pharm Res*, and eight other collaborative works. Won American Physiological Society Caroline tum Suden Award, APS and The University of Chicago Robert Priest Award.

2006-10 David Raleigh, M.D.,Ph.D./Research and Clinical Fellow in Radiation Oncology, University of California San Francisco
Medical Scientist Training Program (MSTP) Student who studied tight junction associated marvel proteins in terms of structure, function, and molecular dynamics, published first author papers in *J Cell Biol* and *Mol Biol Cell* and five other collaborative works. Was awarded multiple abstract prizes.

2006-08 Tiffany Booker/Math Instructor, Noble Auburn Gresham Charter School, Chicago, IL
Undergraduate Student supported by NIH Minority Supplement who studied myosin light chain kinase in diarrheal disease. Later joined Teach for America before current position.

2007-08 Harry Rosenberg/M.D.-Ph.D. Student, University of Illinois
Undergraduate Student who studied myosin light chain kinase trafficking. Won Best Undergraduate Honors Thesis award. After graduation from The University of Chicago matriculated into M.D.-Ph.D. program at the University of Illinois

2007-08 Aaron Hecht/M.D.-Ph.D. (MSTP) Student, The University of Chicago
Undergraduate Student awarded Foundation for Digestive Health (American Gastroenterological Association) Research Fellowship and studied mechanisms of

intestinal wound closure in vivo. After graduation from Washington University, St. Louis, matriculated into M.D.-Ph.D. program at The University of Chicago.

2007-09 Erika Sullivan, M.D., M.S./Assistant Professor (Clinical), University of Utah
M.S. student who studied microbial interactions with the tight junction. Subsequently received M.D. from The University of Chicago and completed family medicine residency at the University of Utah.

2007-16 Christopher Weber, M.D., Ph.D./Assistant Professor, Department of Pathology, The University of Chicago
Postdoctoral Fellow and Faculty member who studied tight junction conductance pathways in vitro and in vivo. Published three first author papers (*J Biol Chem, Lab Invest, eLife), one co-senior author paper (Mol Biol Cell*), a first author review in *Annual Review of Physiology*, and 15 other first author and collaborative works. Finalist in Burroughs Wellcome Career Awards for Medical Scientists. Awarded F32, K08, and R03 from NIH. First author work was selected for presentation at the Basic Science Plenary Session at Digestive Disease Week 2013.

2008, 2009, 2010, 2012-13 Devin Boe/M.D.-Ph.D. Student, Loyola University (Chicago)
Undergraduate Student (summer research 2008) awarded a Foundation for Digestive Health (American Gastroenterological Association) Research Fellowship (2009) and a Crohn's & Colitis Foundation of America Research Fellowship (2010) who also worked as a Research Assistant after graduation (2012-13) studying occludin trafficking and barrier regulation. After graduation from Northwestern University, matriculated into M.D.-Ph.D. program at Loyola University (Chicago).

2008-10 Jingshing Wu, Ph.D./M.D. Student, The University of Chicago
Medical Student who studied myosin light chain kinase trafficking. Subsequently took leave of absence to complete a Ph.D. in cell biology at Yale before returning to medical school at The University of Chicago.

2008-15 Karen Edelblum, Ph.D./Assistant Professor and Chancellor Scholar, Department of Pathology & Laboratory Medicine, Center for Inflammation and Immunity, Rutgers New Jersey Medical School
Postdoctoral Fellow and Faculty member who studied intraepithelial lymphocyte migration and function. Supported by a Research Scholar Award from Foundation for Digestive Health (American Gastroenterological Association), F32, K01, and R03 grants from NIH, and local grants from both The University of Chicago Digestive Disease Center and the Gastrointestinal Research Foundation (GIRF). Published first author papers in *PNAS* and *Gastroenterology*, a first author review in *Curr Opin Pharmacol*, a first author book chapter in textbook of Mucosal Immunology, and several additional collaborative works.

2009 Sherry Fu
High School Student (summer research) supported by Foundation for Digestive Health (American Gastroenterological Association) Research Fellowship who studied myosin light chain kinase trafficking. Subsequently received undergraduate degree from MIT.

2009-10 Markus Gluth, Ph.D., M.S./Postdoctoral Fellow, Humboldt-Universität zu Berlin,

Germany
Visiting Ph.D. Student who studied mechanisms of tight junction regulation.

2009-11 Benjamin Umans, M.S./Ph.D. student, Biological and Biomedical Sciences, Harvard University
Undergraduate student and Research Assistant who studied ZO-1 dependent mechanisms of tight junction regulation. After receiving undergraduate degree from The University of Chicago was awarded Marshall Fellowship at Cambridge University and then entered Ph.D. program at Harvard Medical School.

2010-12 Nora Joseph, M.D./Attending Pathologist, NorthShore University Health System
Postdoctoral Fellow who studied contributions of ZO-1 to epithelial morphogenesis and is a co-author on three papers (*Sci Transl Med, Cancer Res, Arthritis Rheum).*

2010 Hannah Weinberg-Wolf/Ph.D. Student, Yale University
High School Student (summer research) supported by Foundation for Digestive Health (American Gastroenterological Association) Research Fellowship who studied myosin light chain kinase trafficking. Subsequently received undergraduate degree from Johns Hopkins.

2010 Emily S. Turner/Director's Financial Analyst, Office of the Director, Consumer Financial Protection Bureau
High School Student who studied occludin tail structure. Published one first author commentary in *Gastroenterology.* Subsequently received undergraduate degree from Carleton College.

2010-12 Emily Bradford, Ph.D./Postdoctoral Fellow, University of Kentucky
Postdoctoral Fellow who studied myosin light chain kinase function and was supported by a F32 grant from NIH. Published collaborative works in *J Clin Invest*, *J Cell Biol*, and *Amer J Physiol – Cell Physiol* and a first author book chapter.

2011 Stefania Vetrano, M.D., Ph.D./Staff Scientist, Istituto Clinico Humanitas, Milan, Italy
Visiting Scientist who studied barrier function in colitis.

2011 Susanne Krug, Ph.D./Assistant Professor (equivalent), Institute of Clinical Physiology, Charitè, Campus Benjamin Franklin, Berlin, Germany
Visiting Scientist who worked to develop of transgenic mice expressing fluorescent tight junction proteins.

2011 Kevin Cunningham, Ph.D./Pre-Clinical Assessor, Irish Medicines Board, Dublin, Ireland
Postdoctoral Fellow who studied claudin-2 expression and function. Training was cut short by a family crisis in Ireland.

2011 Kenny Anderson/M.S. Student, Western Kentucky University
Undergraduate student who studied occludin function in TNF-induced tight junction regulation and is a co-author on a publication in *Mol Biol Cell.*

2011, 2012 Stephanie Schmitt/M.D. Student, Rush University School of Medicine, Chicago, IL

Undergraduate student who studied occludin function in TNF-induced tight junction regulation and was supported by a Foundation for Digestive Health (American Gastroenterological Association) Research Fellowship. After graduation from Wellesley College, matriculated into medical school at Rush University School of Medicine, Chicago.

2011-13 Harsha Rajapakse, Ph.D./Visiting Assistant Professor of Chemistry, St. John's College, CUNY, Jamaica, New York
Postdoctoral Fellow who studied mechanisms of claudin function and is a co-author on a publication in *Mol Biol Cell*.

2011-12 Heewon Aimee Kwak, M.D./Chief Resident, Department of Pathology, The University of Chicago
Postdoctoral Fellow who studied barrier function in graft vs. host disease and is a co-author on one paper in *Sci Transl Med*. After research experience entered pathology residency program at The University of Chicago.

2011-14 Mary Buschmann, Ph.D./Research Manager, The University of Chicago
Postdoctoral Fellow who studied the role of occludin in TNF-induced barrier loss, published one first author paper in *Mol Biol Cell*, and was supported by a F32.

2011-15 Matthew Odenwald, Ph.D./M.D. Student, The University of Chicago
M.D.-Ph.D. Student who studied contributions of ZO-1 to epithelial morphogenesis. Received NIH F30 and The University of Chicago Robert Priest Award.

2011-15 Amulya Lingaraju/Ph.D. Student, The University of Chicago
Research Assistant who studied the role of occludin in epithelial damage. Subsequently joined Ph.D. program at The University of Chicago.

2011-14 Ekaterina Khramtsova, Ph.D./Postdoctoral Fellow, The University of Chicago
Ph.D. Student who studied claudin-2 function and was a co-author on three publications. First author work was selected for presentation at the Basic Science Plenary Session at Digestive Disease Week 2014.

2012-13 Lydia Breskin/M.D. Student, Tulane School of Medicine, New Orleans, LA
Research Assistant who studied tight junction associated Marvel proteins in colitis.

2012-14 Juanmin Zha, M.D./Attending endocrinologist, Nanjing, China
Postdoctoral Fellow who studied myosin light chain kinase and phosphatase functions and is a co-author on a publication in *Mol Biol Cell*.

2012-15 Weiqi He, Ph.D./Professor, Cambridge-Suda (CAM-SU) Genomic Resource Center, Soochow University, Suzhou, China
Postdoctoral Fellow who studied myosin light chain kinase and phosphatase functions in colitis and was supported by a Crohn's and Colitis Foundation of America Research Fellowship.

2012-15 Pei-Yun Tsai, Ph.D.

Postdoctoral Fellow who studied claudin-2 function in disease and was a co-author on one publication in *Gastroenterology*.

2013 Zachary Smith/Undergraduate Student, Vanderbilt University, Nashville, TN
Summer Student who studied mechanisms of ZO-1 function and was supported by a Foundation for Digestive Health (American Gastroenterological Association) Research Fellowship.

2013 Elliot Abbott, M.Sc./Applying to medical school
Summer Student who studied mechanisms of claudin function and is a co-author on a publication in *Mol Biol Cell* and was supported by a Crohn's and Colitis Foundation of America Summer Research Fellowship. After graduation from the Unioversity of Florida earned M.Sc. in radiation biology at Oxford, UK.

2013 Jonathan King, Ph.D./Associate Professor and Chair, Department of Biology, Trinity University, San Antonio, TX
Visiting Scientist who worked on domain-specific ZO-1functions.

2013-14 Johannes Reiner, M.D. Student, Eberhard Karls University, Tubingen, Germany
Medical Student who studied mechanisms of claudin function and was supported by a Böhringer Ingelheim Foundation Research Fellowship

2013-14 Bingkun Zhang, Ph.D./Associate Professor, State Key Laboratory of Animal Nutrition, Department of Animal Nutrition & Feed Science, College of Animal Science & Technology, China Agricultural University, Beijing, China
Postdoctoral Fellow who studied claudin-2 function.

2013-15 Michael Warren/Undergraduate Student, The University of Chicago
Undergraduate Student who studied occludin structure and was supported by Foundation for Digestive Health (American Gastroenterological Association) Research Fellowship (2013), American Physiological Society Summer Research Fellowship (2014), and the Katen Scholars Program (2015).

2013-15 Erin McAuley/Ph.D. Student, The University of Chicago
Ph.D. Student who studied myosin light chain kinase trafficking, was a co-author on one publication in *Mol Biol Cell*, and was awarded an F31. Training was terminated prematurely and the award was declined due to an inability to relocate with the lab to Boston.

2013-15 Sheng-Ry Shiou, Ph.D.
Postdoctoral Fellow who studied claudin-2 function in disease.

2013- Catherine Hunter, M.D./Assistant Professor of Pediatrics (Surgery), Feinberg School of Medicine, Northwestern University
Faculty Mentee who studies necrotizing enterocolitis and was supported by a Research Scholar Award from Foundation for Digestive Health (American Gastroenterological Association) and a K08 from the NIH.

2015-	Marion France, Ph.D./Postdoctoral Fellow, Turner lab Postdoctoral Fellow studying cell biology of claudin proteins.
2015-	Sunil Yeruva, Ph.D./Postdoctoral Fellow, Turner lab Postdoctoral Fellow studying claudin-2 function in disease.
2015-16	Jeremy R. Herrmann/Visiting Medical Student, Turner lab Visiting Medical Student who studied barrier and transport functions in pediatric disease.
2015-	Aaron Buckley/Research Assistant, Turner lab Research Assistant studying MLCK1 protein interactions.
2016-	Wangsun Choi, Ph.D./Postdoctoral Fellow, Turner lab Postdoctoral Fellow studying MLCK1 trafficking in disease.
2016	Sana Syed, M.D., M.Sc./ Advanced Nutrition Fellow, Boston Children's Hospital Fellow who spent 1 month learning methods of high throughput tissue analysis in the context of environmental enteropathy.
2106-	Nitesh Shashikanth, Ph.D. /Postdoctoral Fellow, Turner lab Postdoctoral Fellow studying occludin/ZO-1 interactions and mechanotransduction at the tight junction.
2016-	Wei-Ting Kuo, Ph.D. /Postdoctoral Fellow, Turner lab Postdoctoral Fellow studying ZO-1 function in epithelial responses to injury.

Formal Teaching of Peers (e.g., CME and other continuing education courses)

1996-99	Weekly Gastrointestinal Oncology Tumor Board. Practicing and Training Surgeons, Oncologists, Radiologists, and Pathologists	Barbara Ann Karmanos Cancer Institute. Presenter and Discussant. 1 hour of direct contact each week.
1996-01	Weekly Gastroenterology Conference. Practicing and Training Gastroenterologists	Detroit Medical Center and Harper Hospital Presenter and Discussant. 1 hour of direct contact each week.
2001-11	Inflammatory Bowel Disease Conference. Practicing and Training Gastroenterologists, Surgeons, and Pathologists m	The University of Chicago Presenter and Discussant. 2 hours of direct contact each month.
2003	Update in Surgical Pathology CME course. Barrett's esophagus: Location, location, location Practicing Pathologists	The University of Chicago Lecturer, 1 hour of direct contact
2003	Update in Surgical Pathology CME course. Diagnosis and management of diarrheal disease: Flow of information is key	The University of Chicago

	Practicing Pathologists	Lecturer, 1 hour of direct contact
2005	Update in Surgical Pathology CME course. How do I decide if it is really colitis? Practicing Pathologists	The University of Chicago Lecturer, 1 hour of direct contact
2005	Update in Surgical Pathology CME course. Diagnosis and management of dysplasia in inflammatory bowel disease Practicing Pathologists	The University of Chicago Lecturer, 1 hour of direct contact
2008	Annual Educational Conference: Diagnostic and Management Issues in Inflammatory Bowel Disease Practicing Gastroenterologists	Illinois Crohn's & Colitis Foundation of America, Chicago, IL Lecturer, 1 hour of direct contact
2009	Update in Surgical Pathology CME course. Diagnosis and management of dysplasia in IBD: What happens after I call it? Practicing Pathologists	The University of Chicago Lecturer, 1 hour of direct contact
2016	Gastrointestinal, Liver, and Pancreatic Pathology. Pathogenetic Mechanisms in Inflammatory Bowel Disease. Practicing Pathologists	CME course offered by Brigham and Women's Hospital, Massachusetts General Hospital, and Harvard Medical School Lecturer, 1 hour of direct contact

Local Invited Presentations

No presentations below were sponsored by outside entities

1995	Invited Speaker	Na^+-glucose cotransport-dependent regulation of intestinal epithelial tight junctions	Harvard Digestive Disease Center. Harvard Medical School. Boston, MA.
1996	Invited Speaker	Regulation of Epithelial Tight Junctions: Molecular Mechanisms.	Department of Physiology. Wayne State University School of Medicine. Detroit, MI.
1999	Invited Speaker	Intracellular signaling pathways on the intestine: The sweet story of Na^+-glucose cotransport dependent tight junction regulation.	Institute of Chemical Toxicology. Wayne State University, Detroit, MI.
1999	Invited Speaker	'Putting the squeeze' on the tight junction: Understanding cytoskeletal regulation.	Department of Pathology. Wayne State University School of Medicine. Detroit, MI.
2002	Invited Speaker	'Putting the squeeze' on the tight junction: Understanding cytoskeletal regulation.	Department of Pediatrics, University of Chicago, Chicago, IL

2002	Invited Speaker	Intestinal permeability in inflammatory bowel disease.	Department of Medicine, Section of Gastroenterology-Nutrition, University of Chicago, Chicago, IL
2002	Invited Speaker	Intestinal tight junction regulation: How sweet it is.	Committee on Cell Physiology, University of Chicago, Chicago, IL
2002	Invited Speaker	Actomyosin regulation of gates and fences.	Committee on Molecular Medicine, University of Chicago, Chicago, IL
2002	Invited Speaker	MLCK sets a PIK for the tight junction.	Channels, Drugs, and Cells Seminar Series, University of Chicago, Chicago, IL
2003	Invited Speaker	Coordination of epithelial transport, barrier function, and wound repair: Mechanisms and novel therapeutic approaches.	Department of Neurobiology, Pharmacology and Physiology Annual Retreat. University of Chicago, Chicago, IL
2008	Invited Speaker	Barrier dysfunction in inflammatory bowel disease: Here's leaking at you, kid.	Digestive Diseases Research Core Centers (DDRCC) Retreat. Chicago, IL
2009	Invited Speaker	Tight junction regulation: the role of myosin light chain kinase.	University of Chicago Celiac Disease Center. Chicago, IL

Report of Regional, National and International Invited Teaching and Presentations

No presentations below were sponsored by outside entities

Invited Presentations and Courses

Regional

1998	Visiting Professor	Na^+-glucose cotransport-dependent regulation of intestinal epithelial tight junctions: The role of myosin light chain.	Department of Microbiology, Michigan State University. East Lansing, MI.
1998	Invited Speaker	GI Pathology: Things you wanted to know about, but were afraid to ask, or Evaluation of inflammatory, metaplastic, and dysplastic lesions of the gastroesophageal junction: The pathologist's perspective.	Gastroenterology and Hepatology: Advances in Diagnosis and Treatment. Mackinac, MI
1999	Invited Speaker	Na^+-glucose cotransport-dependent tight	Department of Medicine, Division of Gastroenterology,

		junction regulation: How sweet it is.	Gut Peptide Center. The University of Michigan. Ann Arbor, MI.
1999	Invited Speaker	Regulation of intestinal barrier function in health and disease.	Gastroenterology Research Conference. William Beaumont Hospital. Royal Oak, MI.
2002	Invited Speaker	Setting a PIK for the tight junction.	Chicago Cytoskeleton. Northwestern University School of Medicine, Chicago, IL
2003	Visiting Professor	Maintenance of the epithelial barrier at mucosal surfaces: actomyosin-dependent tight junction function.	Section of Digestive Diseases and Nutrition. The University of Illinois at Chicago. Chicago, IL
2003	Visiting Professor	Maintenance of the epithelial barrier at mucosal surfaces: actomyosin-dependent regulation transport functions.	Division of Nutritional Sciences. The University of Illinois at Urbana-Champaign. Urbana, IL
2005	Visiting Professor	Eliminating the static: Tight junction dynamics exposed.	Department of Medicine (Pulmonary and Critical Care), Northwestern University School of Medicine. Chicago, IL
2005	Visiting Professor	Pathophysiology of mucosal barrier function (Do ya feel leaky? Well do ya…?)	Department of Microbiology. Medical College of Wisconsin. Milwaukee, WI.
2006	Visiting Professor	The mucosal barrier in disease: All in all it's just another break in the wall.	Grand Rounds. Department of Medicine. University of Illinois at Chicago. Chicago, IL
2007	Invited Speaker	'Arrowsmith' or 'Caps for Sale': Choices for the physician-scientist.	Graduation Seminar and Banquet. MD/PhD Training Program. University of Illinois at Chicago.
2007	Visiting Professor	Overcoming barriers in understanding of mucosal disease.	Department of Cell Biology, Neurobiology, and Anatomy. Loyola University Medical Center. Maywood, IL
2007	Invited Speaker	Pathobiology of inflammatory bowel disease: Does form follow function?	Evanston-Northwestern Hospital. Evanston, IL
2008	Invited Speaker	Mechanisms of Epithelial Barrier Regulation: Here's leaking at you, kid.	Northwestern University Medical School. Department of

			Pathology. Chicago, IL
2008	Visiting Professor	Tight junction regulation: No static at all.	Department of Cell Biology. Medical College of Wisconsin. Milwaukee, WI.
2009	Visiting Professor	Mechanisms of barrier regulation: No static at all.	Grand Rounds. Department of Medicine. University of Illinois-Chicago. Chicago, IL
2009	Invited Speaker	Looking at leaking: New views of epithelial barrier regulation and repair.	Chicago Biomedical Research Consortium "Seeing small is believing big." Chicago. Chicago, IL
2010	Invited Speaker	"I'm fixing a hole..." Novel approaches to drugging the mucosal barrier.	Chicago Crosstown Digestive Diseases Research Club
2013	Distinguished Lecturer	A Mechanistic Approach to Treating the Mucosal Barrier	Grand Rounds, Lurie Children's Hospital, Chicago, IL
2013	Visiting Professor	The intestinal epithelial barrier: A new therapeutic target in graft vs. host disease?	Lurie Children's Hospital, Chicago, IL
2014	Visiting Professor	Plasticity of mucosal barriers: From pathogenic mechanisms to therapeutic exploitation.	Northwstern University School of Medicine, Chicago, IL
2016	Speaker	The yin and yang of mucosal barriers.	Research Conference, Department of Pathology, Brigham and Women's Hospital, Boston, MA.
National			
1995	Invited Speaker	Regulation of intestinal barrier function in health and disease	Department of Pathology. Johns Hopkins University School of Medicine. Baltimore, MD.
1995	Invited Speaker	Regulation of intestinal barrier function in health and disease	Department of Pathology, Wayne State University School of Medicine, Detroit, MI.
1999	Invited Speaker	"Putting the squeeze" on the tight junction: Understanding cytoskeletal regulation	Department of Medicine, Division of Gastroenterology, Inflammatory Bowel Disease Research Center, University of Chicago, Chicago, IL

2000	Invited Speaker	Show me the pathway! Mechanisms of signal transduction in intestinal permeability	Department of Pathology, University of Illinois School of Medicine, Chicago, IL
2000	Invited Speaker	"Putting the squeeze" on the tight junction: Exploring molecular mechanisms	Department of Pathology, Dartmouth Medical School, Hanover, NH.
2001	Invited Speaker	"Putting the squeeze" on the tight junction: Understanding cytoskeletal regulation	Department of Pathology, University of Chicago, Chicago, IL
2001	Invited Speaker	Sometimes things don't look so bad	Diagnostic Gastrointestinal Pathology Seminar. Department of Pathology, University of Chicago, Chicago, IL
2001	Invited Speake	Tight junction regulation: Sometimes you get what you NHEed	Department of Pathology, University of Washington, Seattle, WA.
2001	Invited Speaker	Sometimes things don't look so bad	Department of Pathology, University of Washington, Seattle, WA
2001	Visiting Professor	"Putting the squeeze" on the tight junction: Understanding cytoskeletal regulation	Department of Pathology, Case Western Reserve University, Cleveland, OH
2001	Visiting Professor	Sometimes things don't look so bad	Diagnostic Gastrointestinal Pathology Seminar. Department of Pathology, Case Western Reserve University, Cleveland, OH
2001	Invited Speaker	Tight junction regulation: Sometimes you get what you NHEed	Gastrointestinal Tract IX. FASEB Summer Research Conference. Kalispell, MT
2002	Visiting Professor	Setting a PIK for the epithelial barrier: Tight junction regulation as a therapeutic target	Department of Pathology, Emory University, Atlanta, GA
2002	Invited Speaker	Setting a PIK for the epithelial barrier: A novel therapeutic strategy	Central Society for Clinical Research Annual Meeting, Chicago, IL
2003	Visiting Professor	Maintenance of the epithelial barrier at mucosal surfaces: actomyosin-	Gastroenterology Grand Rounds. Boston University Medical

		dependent regulation of cell-cell interactions	Center, Boston, MA
2003	Visiting Professor	Maintenance of the epithelial barrier at mucosal surfaces: actomyosin-dependent tight junction regulation	Harvard Digestive Disease Center. Boston, MA
2003	Visiting Professor	Chronic diarrhea: Many diagnoses, few clues	Department of Medicine (Gastroenterology), The University of New Mexico, Albuquerque, NM
2003	Visiting Professor	Coordination of epithelial transport, barrier function, and wound repair: Mechanisms and novel therapeutic approaches	Department of Medicine (Gastroenterology), The University of New Mexico, Albuquerque, NM.
2004	Meet the Professor Speaker	Tight junction biology in intestinal disease	American Gastroenterological Association 2004 Annual Meeting, New Orleans, LA
2004	Invited Speaker	The function of brush cells in the gastrointestinal tract	NHLBI Workshop on Brush Cell Function. Bethesda, MD Published in Am J Respir Crit Care Med. 2005. 172: 136-9.
2004	Invited Speaker	Plugging the holes: The roles of the tight junction and cytoskeleton in maintenance of mucosal barriers in IBD	Northwest Chapter Crohn's & Colitis Foundation of America. Seattle, WA
2005	Invited Speaker	Dynamics of tight junction structure	Gastrointestinal Tract XI. Innovations in GI Research and Therapy. FASEB Summer Research Conference. Snowmass, CO
2005	Visiting Professor	Pathophysiology of mucosal barrier function	Department of Pathology, Immunology, and Laboratory Medicine, University of Florida, Gainesville, FL
2005	Visiting Professor	Lumps, bumps, and nodules: Diagnostic challenges in gastrointestinal pathology	Department of Pathology, Immunology, and Laboratory Medicine, University of Florida. Gainesville, FL
2005	Visiting Professor	Diagnosis and management of dysplasia in inflammatory bowel disease	Department of Pathology, Immunology, and Laboratory Medicine, University of Florida.

			Gainesville, FL
2005	Invited Speaker	State of the art lecture: Healing the intestinal epithelial barrier: potential for new therapies	Crohn's & Colitis Foundation of America National Research and Clinical Conference - 4th Annual Advances in the Inflammatory Bowel Diseases. Miami Beach, FL
2005	Visiting Professor	Pathophysiology of mucosal barrier function: Cytoskeletal regulation of tight junctions	Department of Medicine (Gastroenterology) and Center for Epithelial Disorders. Johns Hopkins University School of Medicine. Baltimore, MD
2006	Visiting Professor	Pathophysiology of mucosal barrier function in inflammatory bowel disease	Department of Medicine (Gastroenterology) and Digestive Health Center of Excellence. University of Virginia. Charlottesville, VA
2006	Visiting Professor	Cytoskeletal Regulation of the Mucosal Barrier: Setting a PIK for the Tight Junction	Department of Physiology. University of Tennessee. Memphis, TN
2006	Visiting Professor	The Mucosal Barrier in Disease: All in all it's just another break in the wall	Department of Pathology. Emory University. Atlanta, GA
2006	Visiting Professor	Lumps, bumps, and nodules: Diagnostic challenges in gastrointestinal pathology	Diagnostic Pathology Seminar. Department of Pathology. Emory University. Atlanta, GA
2006	Invited Speaker	Eliminating the static: tight junction dynamics exposed	American Physiological Society Symposium "Role of epithelial cells in initiation and propagation of intestinal inflammation" Experimental Biology. San Francisco, CA
2006	Awardee	Molecular basis of epithelial barrier regulation: From basic science to clinical application	American Society for Investigative Pathology Amgen Award Lecture. Experimental Biology. San Francisco, CA
2006	Visiting Professor	The leaky intestinal barrier: A common factor in inflammatory and infectious disease?	West Virginia University Center for Immunopathology & Microbial Pathogenesis, Morgantown, WV

2006	Invited Speaker	The leaky epithelial barrier in intestinal disease	NIAAA Symposium on Alcohol, Intestinal Bacterial Growth, Intestinal Permeability to Endotoxin, Medical Consequences, and Dietary Supplements. Bethesda, MD
2006	Visiting Professor	.The leaky epithelial barrier in intestinal disease	Warren Medical Research Center for Celiac Disease. University of California, San Diego. La Jolla, CA
2006	Invited Speaker	State of the art lecture: Repair and healing of the intestinal epithelium and mucosa	Crohn's & Colitis Foundation of America National Research and Clinical Conference - 5th Annual Advances in the Inflammatory Bowel Diseases. Miami Beach, FL
2007	Visiting Professor	The mucosal barrier in intestinal disease: just another break in the wall?	GI Research Conference. Vanderbilt University. Nashville, TN
2007	Visiting Professor	Lumps, bumps, and nodules: Diagnostic challenges in gastrointestinal pathology	Diagnostic Pathology Seminar. Vanderbilt University. Nashville, TN
2007	Visiting Professor	The mucosal barrier in disease: All in all it's just another break in the wall	Department of Pathology. Case Western Reserve University. Cleveland, OH
2007	Visiting Professor	The mucosal barrier in disease: All in all it's just another break in the wall	Department of Physiology. University of Texas Southwestern Medical Center. Dallas, TX
2007	Invited Speaker	The mucosal barrier in intestinal disease: All in all it's just another break in the wall	Gordon Conference on Salivary Glands and Exocrine Secretion. Ventura, CA
2007	Invited Speaker	The dynamic tight junction during injury	American Gastroenterological Association Research Symposium "Enterocyte signaling during injury and repair" Washington, DC
2007	Invited Speaker	Do ya feel leaky?, well do ya?	ALBA Therapeutics Scientific Advisory Board meeting.

			Baltimore, MD
2007	Visiting Professor	The mucosal barrier in intestinal disease: just another break in the wall?	Department of Medicine. University of Michigan. Ann Arbor, MI
2007	Invited Speaker	Mucosal barrier dysfunction in IBD: Cause, effect, or both?	Mount Sinai School of Medicine. New York, NY
2008	Visiting Professor	The mucosal barrier in disease: Do ya feel leaky?	University of Pennsylvania. Division of Gastroenterology. Philadelphia, PA
2008	Visiting Professor	The mucosal barrier in intestinal disease: Just another break in the wall?	Harvard Medical School Pathology Grand Rounds. Departments of Pathology, Harvard Medical School and Brigham and Women's Hospital. Boston, MA`
2008	Visiting Professor	Overcoming barriers to understanding mucosal disease	Yale University. Department of Pathology. New Haven, CT
2008	Visiting Professor	Diagnostic Conference: Lumps and bumps – Problems in GI Pathology.	Yale University. Department of Pathology. New Haven, CT
2008	Visiting Professor	Mucosal barrier dysfunction and immune activation in IBD: A chicken or egg paradox	Cincinnati Children's Hospital Medical Center. Digestive Health Center. Cincinnati, OH
2008	Invited Speaker	Mechanisms and implications of epithelial barrier regulation: Here's leaking at you, kid	Washington University Medical School. Departments of Pathology and Medicine (Gastroenterology). St. Louis, MO
2008	Invited Speaker	The role of myosin in gut permeability: Here's leaking at you, kid	Cytokinetics. South San Francisco, CA
2008	Invited Speaker	What role does intestinal permeability and epithelial cell barrier function play in IBD? Here's leaking at you, kid.	Crohn's & Colitis Foundation National Research & Clinical Conference (CCFA). Hollywood, FL
2009	Invited Speaker	Cytoskeletal mechanisms of barrier regulation	American Gastroenterological Association Research Symposium "Mechanisms and impact of barrier dysfunction in intestinal disease" Chicago, IL

2009	Visiting Professor	Mechanisms and impact of barrier regulation: No static at all	National Institutes of Health (NIAID). Bethesda, MD
2009	Invited Speaker	Pathologies of the GI tract	ICMI 2009 Pre-Congress Workshop: Microbes and Mucosal Immunity. Boston, MA
2009	Invited Speaker	Tight junction structure and regulation: No static at all	FASEB Summer Research Conference "Advances in the molecular and cell biology of the intestinal epithelium." Snowmass Village, CO
2009	Invited Speaker	Tight junctions and epithelial function: No static at all	Saban Research Institute and Department of Surgery. University of Southern California and Children's Hospital of Los Angeles. Los Angeles, CA
2009	Invited Speaker	Molecular anatomy of the tight junction: No static at all	Symposium entitled: "Tight junctions in kidney health and disease" American Society of Nephrology Annual Meeting. San Diego, CA
2009	Visiting Professor	Tight junction structure and regulation: No static at all	Department of Pharmacology. Columbia University College of Physicians and Surgeons. New York, NY
2010	Investigator/Speaker	Healing the intestinal epithelial barrier: Potential for new therapies	Broad Medical Research Program Investigators Meeting. Los Angeles, CA
2010	Visiting Professor	Mucosal barrier function: Mechanisms and implications	Department of Pathology. University of Wisconsin. Madison, WI
2010	Invited Speaker	Role of epithelial tight junctions in initiation and propagation of intestinal inflammation	Featured Topic Symposium "Epithelial barrier function in inflammatory bowel diseases." American Physiological Society Annual Meeting (Cell and Molecular Physiology Section) Anaheim, CA
2010	Invited Speaker	Molecular mechanisms of tight junction	University of California. Los

		regulation: No static at all	Angeles, CA
2010	Invited Speaker	Immunopathologies of the human gastrointestinal tract	Microbes and Mucosal Immunity. Scottsdale, AZ
2010	Invited Speaker	Dysregulation of apical junctional complexes and inflammation	Gastrointestinal Response to Injury. Scottsdale, AZ
2010	Invited Speaker	Transport and immune consequences of barrier defects	Bill & Melinda Gates Foundation Symposium on Gut Integrity. Seattle, WA
2011	Invited Speaker	All barriers are not created equal: molecular regulation of tight junction selectivity	Emory University. Atlanta, GA
2011	Invited Speaker	Intestinal permeability in health and disease: Here's leaking at you, kid	Tulane Primate Center. Covington, LA
2011	Invited Speaker	Cellular and molecular mechanisms of barrier function in relation to stress in IBD and IBS	American Gastroenterological Association Research Symposium "Barrier Function and Intestinal Homeostasis" Chicago, IL
2011	Visiting Professor	Tight junction molecular architecture: modeling of dynamic protein interactions and barrier regulation	Center for Cell Analysis & Modeling. University of Connecticut Health Center Farmington, CT
2011	Invited Speaker	Approaches to and outcomes of barrier restorative therapy. Has the tight junction come of age a druggable target?	NIDDK Workshop "Translational Approaches for Pharmacotherapy Development for Acute Diarrhea." Bethesda, MD
2011	Visiting Professor	A mechanistic approach to treating the mucosal barrier	Cincinnati Children's Hospital Medical Center. Digestive Health Center. Cincinnati, OH
2012	Keynote Speaker	Regulation of mucosal barrier function: A complex problem	University of Michigan. Michigan Gut Peptide Center Retreat. Ann Arbor, MI
2012	Investigator/Speaker	"A novel approach to barrier restoration: More than a diversion."	Broad Medical Research Program Investigator Meeting. Los Angeles, CA

2012	Invited Speaker	Of barriers, microbiota, and inflammatory disease: More than just IBD	Intestinal Pathobiology Symposia Retreat. Georgia State University. Atlanta, GA
2012	Visiting Professor	Studies of dynamic remodeling: Mucosal barriers and the practice of pathology	Department of Pathology. University of Washington. Seattle, WA
2012	Invited Speaker	What are the limitations to intepretation by pathologists and gastroenterologists? Common pitfalls.	IBD Advisory Board. CDx Diagnostics, OralCDx Laboratories Suffern, NY
2012	Invited Speaker	Novel targets for molecular therapies	American Gastroenterological Association Research Symposium "Intestinal permeability: What are the tolerances?" San Diego, CA
2012	Invited Speaker	Role of tight junctions in apical trafficking and disease	Research Symposium "Plasma membrane trafficking in polarized cells: What are the similarities and differences?" Johns Hopkins University School of Medicine. Baltimore, MD
2012	Invited Speaker	Genomics, intestinal epithelial barrier dysfunction, and the pathogenesis of IBD	University of Pennsylvania Center for Molecular Studies in Digestive and Liver Diseases Research Symposium "IBD, Functional Genomics and the Intestinal Mucosal Interface." Philadelphia, PA
2012	Daljit and Elaine Sarkaria Lecturer and Visiting Professor	All in all, it's just another break in the wall, or is it? The roles of intestinal barrier loss systemic disease	University of California, Los Angeles. Los Angeles, CA
2012	Digestive Diseases Grand Rounds Lecturer and Visiting Professor	Here's leaking at you kid: A mechanistic approach to treating the mucosal barrier	University of California, Los Angeles. Los Angeles, CA
2012	Pathology Diagnostic	Lumps, bumps, and stumps	University of California, Los

	Case-based Slide Seminar		Angeles. Los Angeles, CA
2012	GI Fellows Teaching Rounds	How do you solve a problem like dysplasia?	University of California, Los Angeles. Los Angeles, CA
2013	Visiting Professor	A pleasant diversion: Therapeutic restoration of the intestinal barrier	Gastroenterology Grand Rounds. Massachusetts General Hospital, Boston, MA
2013	Visiting Professor	Towards solving tight junction structure-function relationships: Here's leaking at you kid	Gastroenterology Research Seminar. Massachusetts General Hospital, Boston, MA
2013	Visiting Professor	Towards solving tight junction structure-function relationships: Here's leaking at you kid	Department of Cell Biology, University of Maryland, College Park, MD
2013	Visiting Professor	The intestinal epithelial barrier: A new therapeutic target in graft vs host disease?	Center For Vascular and Inflammatory Diseases, University of Maryland, Baltimore, MD
2013	Visiting Professor	Applying new concepts: Are we ready to get serious about therapeutic restoration of intestinal barrier function	Department of Microbiology and Physiological Systems, University of Massachusetts Medical School, Worcester, MA
2013	Visiting Professor	The epithelial barrier in IBD: Is it a viable therapeutic target?	Texas A&M Health Science Center. Temple, TX
2013	Visiting Professor	Plasticity of mucosal barriers	The University of Michigan, Ann Arbor, MI
2013	Invited Speaker	How does the genome modulate the intestinal epithelium in IBD?	2013 Advances in Inflammatory Bowel Diseases meeting. Hollywood, FL
2013	Invited Speaker	Understanding barrier function in IBD	2013 Advances in Inflammatory Bowel Diseases meeting. Hollywood, FL
2014	Visiting Professor	Mechanistic approaches to mucosal barrier restoration and therapeutic intervention	Mayo Clinic, Rochester, MN
2014	Invited Speaker	Plasticity of Mucosal Barriers: From Mechanisms to Therapeutic Exploitation	NIH/NICHD, Bethesda, MD

2014	Keynote Speaker	Physiology of the Intestinal Barrier: From Mechanisms to Therapeutic Exploitation	Controlled Release Society, Annual Meeting, Chicago, IL
2014	Distinguished Research Seminar Speaker	Epithelial Barriers: Physiology, Pathobiology and Opportunities for Intervention	Oklahoma Medical Research Foundation, Oklahoma City, OK
2014	Invited Speaker, Frontiers in Digestive Health City-wide Grand Rounds	Plasticity of Epithelial Barriers: Mechanisms and Therapeutic Opportunities	Case Western Reserve University, Cleveland, OH
2014	Invited Speaker	Regulation of mucosal barrier function: Mechanisms and therapeutic opportunities	Brigham and Women's Hospital/Harvard Digestive Disease Center, Boston, MA
2015	Invited Speaker	Tight junction permeability: mechanisms and regulation	Gordon Research Conference: Salivary Glands & Exocrine Biology. Galveston, TX
2015	Visiting Professor	Mucosal Barriers: Physiology, Pathobiology, and Opportunities for Intervention	Uniformed Services University of Health Sciences. Bethesda, MD
2015	Hans Ussing Lectureship	Regulation of mucosal barrier function: Beyond the chambers	American Physiolocal Society. Experimental Biology. Boston, MA
2015	Invited Speaker	Tight junction permeability: mechanisms and regulation	Second Shanthi Sitaraman Intestinal Pathobiology Symposium. Georgia State University. Atlanta, GA
2015	Invited Speaker	Mucosal Barriers: Physiology, Pathobiology, and Opportunities for Intervention	Annual Meeting of the International Scientific Association for Probiotics and Prebiotics. Washington, DC
2015	Invited Speaker	A Reviewer's Perspective on K-Awards	Departments of Medicine and Cell and Developmental Biology. University of Michigan. Ann Arbor, MI
2015	Invited Speaker	Plasticity of Mucosal Barriers	Gordon Research Conference: Cell Contact and Adhesion.

			Andover, NH
2015	Invited Speaker	Tight Junctions in Regulation of Epithelial Barrier and Pathogenesis of Crohn's Disease	Gordon Research Conference: Barrier Function of Mammalian Skin. Waterville Valley, NH
2015	Invited Speaker	Claudin-2 as a regulator of the mucosal barrier: Too much, too little, or just enough?	FASEB Summer Research Conference. Gastrointestinal Tract XVI: GI homeostasis, the microbiome and the barrier, development and disease. Steamboat Springs, CO
2016	Pathology Grand Rounds Speaker	Translational cell biology of mucosal barriers	Brigham and Women's Hospital, Boston, MA
2016	Lecturer	Pathogenetic mechanisms in inflammatory bowel disease	Gatrointestinal, Liver, and Pancreatic Pathology. Harvard Continuing Education Course. Fairmont Copley Plaza Hotel. Boston, MA
2016	Invited Speaker	Tight junction regulation during enteric infection: Benefits of increased permeability.	CAMPS Symposium: Recent advances in the structure and function of epithelial tight junctions. American Physiolocal Society. Experimental Biology. San Diego, CA
2016	Invited Speaker	Overcoming barriers	Eleanor Humphreys Scientific Symposium. The University of Chicago. Chicago, IL
2016	Invited Speaker	Tight Junction Barrier Loss: Friend or Foe?	Mucosal Healing of the Intestinal Epithelial Barrier Symposium. New York Academy of Sciences. New York, NY
International			
2002	Visiting Professor	SGLT1 as master regulator of nutrient absorption	The University of Calgary and Alberta Heritage Foundation for Medical Research. Department of Physiology and Biophysics. Alberta, Canada
2002	Invited Speaker	Cytoskeletal regulation of transcellular	Meeting of the European Intestinal Transport Group.

		and paracellular transport	Heiloo. The Netherlands
2004	Invited Speaker	Plugging the holes: The roles of the tight junction and cytoskeleton in maintenance of the mucosal barrier	European Life Sciences Organization 2004 Meeting 2004 and 8th International World Congress of Cell Biology. Nice, France
2006	Visiting Professor	Molecular basis of epithelial barrier regulation: From basic investigation to therapeutic potential	Royal College of Surgeons in Ireland (RCSI), Dublin, Ireland
2007	Invited Speaker	The mucosal barrier in intestinal disease: just another break in the wall?	Gastrointestinal Diseases Research Unit. Queen's University. Kingston, Ontario. Canada
2007	Faculty	Pathologies of the Human Gastrointestinal Tract	Microbes and Mucosal Immunity Course. Montebello, Quebec. Canada
2007	Invited Speaker	The Dynamics of Tight Junction Structure and Function	Gastrointestinal Response to Injury: Conference. Montebello, Quebec. Canada
2008	Invited Speaker	Inflammatory bowel disease: Not just another break in the wall	International conference "Molecular structure and function of the tight junction: From basic mechanisms to clinical manifestations" Berlin, Germany
2009	Invited Speaker	Intestinal Barrier Defects	Falk Symposium-Inflammation in the Intestinal Tract: Pathogenesis and Treatment. Kiev, Ukraine
2009	Visiting Professor and Guest Adjudicator	Molecular basis and implications of mucosal barrier regulation: No static at all	Department of Medicine. University of Alberta. Edmonton, AB
2009	Invited Speaker	A hitch-hiker's guide to the tight junction	Ajinomoto Chemical Co. Kawasaki, Japan
2009	Invited Speaker	Tight junction remodeling in cytoskeletally-mediated barrier regulation: the unique role of ZO-1	IUPS Tight Junction Symposium, XXXVI International Congress of Physiological Sciences. Kyoto, Japan

2009	Visiting Professor	Tight junction remodeling and cytoskeletally-mediated barrier regulation	RIKEN Center for Developmental Biology. Kobe, Japan
2010	Invited Speaker	Tight junction regulation: Molecular mechanisms and roles in pathogenesis	US-Japan GI Executive meeting. Tokyo, Japan
2010	Visiting Professor	Structure-function relationships in epithelial barrier regulation	Osaka University. Osaka, Japan
2010	Invited Speaker	Tight junction regulation: Molecular mechanisms and roles in pathogenesis	Research Symposium "Epithelial tight junctions." Annual Meeting of The Physiological Society (UK). Manchester, UK
2010	Visiting Professor	Structure-function relationships in epithelial barrier regulation	Institut Curie. Paris, France
2010	Invited Speaker	Intestinal mucosal barrier function in health and disease	Argentine Congress of Gastroenterology and Digestive Endoscopy. Buenos Aires, Argentina
2010	Invited Speaker	The contribution of barrier dysfunction to digestive disease: Coordination of paracellular and transcellular transport	Argentine Congress of Gastroenterology and Digestive Endoscopy. Buenos Aires, Argentina
2010	Invited Speaker	Bugs, cytokines, and drugs: Factors that influence the tight junction barrier	Argentine Congress of Gastroenterology and Digestive Endoscopy. Buenos Aires, Argentina
2010	Invited Speaker	Mucosal immunity and intestinal barrier dysfunction: New concepts for the clinical gastroenterologist	Argentine Congress of Gastroenterology and Digestive Endoscopy. Buenos Aires, Argentina
2010	Invited Speaker	Intestinal barrier function: Molecular regulation and disease pathogenesis	"Transepithelial Transport Mechanisms 2010." Copenhagen, Denmark
2011	Invited Speaker	Molecular structure and function of the apical junction complex in health and disease	Canadian Digestive Disease Week meeting. Vancouver, Canada
2011	Invited Speaker	Molecular mechanisms of intestinal epithelial tight junction regulation	Annual Meeting of The Physiological Society (UK). Oxford, UK

2011	Invited Speaker	From tight junction molecular interactions to barrier restorative therapeutic approaches	International conference "Molecular structure and function of the tight junction: From basic mechanisms to clinical manifestations." Berlin, Germany
2011	Invited Speaker	Molecular regulation of the tight junction: A complex problem	Gastrointestinal Research Group. University of Calgary. Calgary, Canada
2011	Lectureship in Inflammatory Bowel Disease	Stopping the flood: The gut barrier and its role in inflammatory bowel disease	Intestinal Disease Research Unit (IDRU), University of Calgary, Canada
2011	Invited Speaker	Molecular Basis of Intestinal Barrier Function: Implications for Immune-Mediated Disease	Keystone Symposium. "Malnutrition, Gut-Microbial Interactions and Mucosal Immunity to Vaccines." New Delhi, India
2012	Visiting Professor	Maintenance of epithelial barriers: From cell biology to pathophysiology and therapeutics	Rostock University Medical School. Rostock, Germany
2012	Invited Speaker	Molecular Mechanisms of Mucosal Barrier Regulation: Implications for Immune-Mediated Disease	5th International Symposium of the SFB 621 Pathobiology of the Intestinal Mucosa. Hannover, Germany
2012	Visiting Professor	Maintenance of epithelial barriers: From cell biology to pathophysiology and therapeutics	University of Geneva. Geneva, Switzerland
2012	Invited Speaker	Towards solving tight junction structure-function relationships: A complex problem	International conference "Molecular Structure and Function of the Apical Junctional Complex in Epithelia and Endothelia." Merida, Mexico
2013	Visiting Professor	A Mechanistic Approach to Treating the Mucosal Barrier.	Sostieni anche tu la Ricerca di Humanitas. Milan, Italy
2013	Visiting Professor	Applying new concepts: Are we ready to get serious about therapeutic function?	Technische Universität München, Munich, Germany
2013	Visiting Professor	The epithelial barrier: What is it, and should we do something about it?	The University of Zambia Lusaka, Zambia

2013	Visiting Professor	Post-translational mechanisms of mucosal barrier regulation: Approaches to therapeutic exploitation	Leibniz-Institut für Molekulare Pharmakologie, Berlin, Germany
2014	Invited Speaker	Mechanistic approaches to mucosal barrier restoration and therapeutic interventions	British Society of Cell Biology/British Society of Developmental Biology Joint Spring Meeting, Warwick, UK
2015	Invited Speaker	Molecular Mechanisms of Claudin Function: Implications for Physiology, Pathobiology, and Therapy	120th Annual Meeting of the Japanese Association of Anatomists and the 92nd Annual Meeting of the Physiological Society of Japan cooperative meeting. Kobe, Japan
2015	Visiting Professor	Regulation of mucosal barrier function: Mechanisms and therapeutic exploitation	National Taiwan University College of Medicine. Taiwan, ROC
2015	Visiting Professor	Role of the intestinal barrier in intestinal and systemic disease: New concepts and opportunities	Taiwan Association for the Study of Small Intestinal Disease. Taiwan, ROC
2015	Visiting Professor	Claudin-2 as a regulator of the mucosal barrier: Too much, too little, or just enough?	National Yang-Min University. Taiwan, ROC
2015	Invited Speaker	Claudin-2 as a regulator of the mucosal barrier: Too much, too little, or just enough?	Symposium: Life with Tight Junctions. Charité, Campus Benjamin Franklin. Berlin, Germany
2016	Invited Speaker	Regulation of mucosal barrier function: Mechanisms and therapeutic exploitation	Centre for Colorectal Disease, St. Vincent's Hospital, Dublin, Ireland
2016	Invited Speaker	Mucosal Barriers: Should We Make Them Great Again?	School of Medicine, Trinity College, Dublin, Ireland
2016	Invited Speaker	Links between intestinal inflammation, barrier function, and inflammatory mediators	International conference "Tight junctions and their proteins." Berlin, Germany

Report of Clinical Activities and Innovations

Current Licensure and Certification

2001-17	Medical License 036104920, State of Illinois,
1994-	Anatomic Pathology, American Board of Pathology (unlimited certification)
2006-16	Voluntary Recertification, American Board of Pathology
2014-24	Voluntary Recertification, American Board of Pathology

Practice Activities

1995-96	Attending Gastrointestinal Surgical Pathologist	Brigham and Women's Hospital, Boston, MA	Approximately 1,000 complex gastrointestinal surgical pathology cases per year
1996-2001	Attending Surgical Pathologist	Harper Hospital and Detroit Medical Center, Detroit, MI	Approximately 1,200 general surgical cases per year. Approximately 300 liver needle biopsies per year. Sole consultant for complex gastrointestinal pathology cases for 5 hospital network.
2001-16	Attending Gastrointestinal Surgical Pathologist	The University of Chicago Hospitals, Chicago, IL	Approximately 1,000 complex gastrointestinal surgical pathology cases per year.
2016-	Attending Gastrointestinal Surgical Pathologist	Brigham and Women's Hospital, Boston, MA	20% effort (8 weeks/year) on the GI Pathology Service (gastrointestinal tract, liver, and pancreas).

Clinical Innovations

Revision of American Joint Committee on Cancer Colon Cancer Staging Guidelines	Made original observation, collected and analyzed cases, and authored 2000 publication by Goldstein and Turner in Cancer. This study shows that it is critical to differentiate between non-lymph node based pericolonic tumor deposits and pericolonic lymph node metastases. The data showed that, non-lymph node pericolonic tumor deposits are a strong risk factor for intraabdominal tumor recurrence. This was particularly true in patients lacking lymph node metastases. The study went on to show that pericolonic tumor deposits are due to perineural or vascular tumor extension. The strength of the data led to revision of the American Joint Committee on cancer colon cancer staging guidelines based on this report.
Advanced understanding of neoplastic risk in ulcerative colitis	Developed novel histologic inflammatory activity scale and analyzed majority of biopsies in a case-control study with 59 cases of ulcerative colitis patients who developed colorectal dysplasia or cancer and 114 control ulcerative colitis patients who did not. Defined risk associated with low grade histologic inflammation and identified protective effect of therapy with select immunomodulatory therapies.
Defined risk of gastrointestinal amyloidosis after hemodialysis	Collected cases, developed and analyzed data, authored manuscript, and was corresponding author of 1998 study published in American Journal of Surgical Pathology showing that risk of developing gastrointestinal β_2-microglobulin amyloidosis correlates with time on dialysis and is particularly increased after 10 years or more of dialysis.

Report of Technological and Other Scientific Innovations

September 29, 2003	Homo sapiens myosin light chain polypeptide kinase isoform 1 (MYLK). mRNA, complete cds, alternatively spliced. From Caco-2 BBe 5E6L. GenBank Accession AY424270; submitted by Clayburgh, D.R. and Turner, J.R.
September 29, 2003	Homo sapiens myosin light chain polypeptide kinase isoform 2 (MYLK). mRNA, complete cds, alternatively spliced. From Caco-2 BBe 5E6L. GenBank Accession AY424269; submitted by Clayburgh, D.R. and Turner, J.R.
May 15, 2006	Homo sapiens MYLK mRNA, 5'UTR, Exons 1B, 2, and part of 3 (translational start site in exon 2). From Caco-2 BBe 5E6L. GenBank Accession DQ642691; submitted by Graham W V., and Turner, J.R.
May 15, 2006	Homo sapiens MYLK mRNA, 5'UTR, Exons 1A, 2, and part of 3 (translational start site in exon 2). From Caco-2 BBe 5E6L. GenBank Accession DQ642692; submitted by Graham W V., and Turner, J.R.
September 8, 2009	Myosin light chain kinase inhibitors and methods of use. US Patent 7,585,844 Inventors: Turner, J.R., and Mrsny, R.J.
September 20, 2009	Homo sapiens myosin light chain kinase (MYLK) gene, promoter region. GenBank Accession GQ981352; submitted by Graham W V., and Turner, J.R.

Report of Scholarship

Peer-Reviewed Publications

Research investigations

1. Seya T, **Turner JR**, and Atkinson JP. Purification and characterization of a membrane protein (gp45-70) that is a cofactor for cleavage of C3b and C4b. J Exp Med. 1986; 163:837-855.
2. **Turner JR**, Tartakoff AM, and Berger M. Intracellular degradation of the complement C3b/C4b receptor in the absence of ligand. J Biol Chem. 1988; 263:4914-4920.
3. **Turner JR** and Tartakoff AM. The response of the Golgi complex to microtubule alterations: the roles of metabolic energy and membrane traffic in Golgi complex organization. J Cell Biol. 1989; 109:2081-2088.
4. **Turner JR**, Tartakoff AM, and Greenspan NS. Cytologic assessment of nuclear and cytoplasmic O-linked N- acetylglucosamine distribution by using anti-streptococcal monoclonal antibodies. Proc Natl Acad Sci U S A. 1990; 87:5608-5612.
5. **Turner JR**, and Tartakoff AM. On the Relation between Distinct Components of the Cytoskeleton - an Epitope Shared by Intermediate Filaments, Microfilaments and Cytoplasmic Foci. Eur J Cell Biol. 1990; 51:259-264.
6. Berger M, Wetzler EM, Welter E, **Turner JR**, and Tartakoff AM. Intracellular sites for storage and recycling of C3b receptors in human neutrophils. Proc Natl Acad Sci U S A. 1991; 88:3019-3023.

‡ Co-corresponding or co-senior author

7. Nusrat A, Giry M, **Turner JR**, Colgan SP, Parkos CA, Carnes D, Lemichez E, Boquet P, and Madara JL. Rho protein regulates tight junctions and perijunctional actin organization in polarized epithelia. Proc Natl Acad Sci U S A. 1995; 92:10629-10633.

8. Probert CS, Chott A, **Turner JR**, Saubermann LJ, Stevens AC, Bodinaku K, Elson CO, Balk SP, and Blumberg RS. Persistent clonal expansions of peripheral blood CD4+ lymphocytes in chronic inflammatory bowel disease. J Immunol. 1996; 157:3183-3191.

9. Tenner S, Carr-Locke DL, Banks PA, Brooks DC, Van Dam J, Farraye FA, **Turner JR**, and Lichtenstein DR. Intraductal mucin-hypersecreting neoplasm "mucinous ductal ectasia": endoscopic recognition and management. Am J Gastroenterol. 1996; 91:2548-2554.

10. **Turner JR**, and Odze RD. Proliferative characteristics of differentiated cells in familial adenomatous polyposis-associated duodenal adenomas. Hum Pathol. 1996; 27:63-69.

11. **Turner JR**, Lencer WI, Carlson S, and Madara JL. Carboxy-terminal vesicular stomatitis virus G protein-tagged intestinal Na+-dependent glucose cotransporter (SGLT1): maintenance of surface expression and global transport function with selective perturbation of transport kinetics and polarized expression. J Biol Chem. 1996; 271:7738-7744.

12. Aster JC, Robertson ES, Hasserjian RP, **Turner JR**, Kieff E, and Sklar J. Oncogenic forms of NOTCH1 lacking either the primary binding site for RBP-Jkappa or nuclear localization sequences retain the ability to associate with RBP-Jkappa and activate transcription. J Biol Chem. 1997; 272:11336-11343.

13. Probert CS, Christ AD, Saubermann LJ, **Turner JR**, Chott A, Carr-Locke D, Balk SP, and Blumberg RS. Analysis of human common bile duct-associated T cells: evidence for oligoclonality, T cell clonal persistence, and epithelial cell recognition. J Immunol. 1997; 158:1941-1948.

14. **Turner JR**, Odze RD, Crum CP, and Resnick MB. MN antigen expression in normal, preneoplastic, and neoplastic esophagus: a clinicopathological study of a new cancer-associated biomarker. Hum Pathol. 1997; 28:740-744.

15. **Turner JR**, Shen LH, Crum CP, Dean PJ, and Odze RD. Low prevalence of human papillomavirus infection in esophageal squamous cell carcinomas from North America: analysis by a highly sensitive and specific polymerase chain reaction-based approach. Hum Pathol. 1997; 28:174-178.

16. **Turner JR**, Rill BK, Carlson SL, Carnes D, Kerner R, Mrsny RJ, and Madara JL. Physiological regulation of epithelial tight junctions is associated with myosin light-chain phosphorylation. Am J Physiol-Cell Ph. 1997; 273:C1378-C1385.

17. Jimenez RE, Price DA, Pinkus GS, Owen WF, Jr., Lazarus JM, Kay J, and **Turner JR**. Development of gastrointestinal beta2-microglobulin amyloidosis correlates with time on dialysis. Am J Surg Pathol. 1998; 22:729-735.

18. Liu L, **Turner JR**, Yu Y, Khan AJ, Jaszewski R, Fligiel SE, and Majumdar AP. Differential expression of EGFR during early reparative phase of the gastric mucosa between young and aged rats. Am J Physiol. 1998; 275:G943-950.

19. Glickman JN, Torres C, Wang HH, **Turner JR**, Shahsafaei A, Richards WG, Sugarbaker DJ, and Odze RD. The prognostic significance of lymph node micrometastasis in patients with esophageal carcinoma. Cancer. 1999; 85:769-778.

20. Saubermann LJ, Probert CS, Christ AD, Chott A, **Turner JR**, Stevens AC, Balk SP, and Blumberg RS. Evidence of T cell receptor beta-chain patterns in inflammatory and noninflammatory bowel disease states. Am J Physiol. 1999; 276:G613-621.

21. Torres C, **Turner JR**, Wang HH, Richards W, Sugarbaker D, Shahsafaei A, and Odze RD. Pathologic prognostic factors in Barrett's associated adenocarcinoma: a follow-up study of 96 patients. Cancer. 1999; 85:520-528.

22. **Turner JR**, Angle JM, Black ED, Joyal JL, Sacks DB, and Madara JL. Protein kinase C-dependent regulation of transepithelial resistance: the roles of myosin light chain and myosin light chain kinase. Am J Physiol. 1999; 277:C554-562.

23. Goldstein NS and **Turner JR**. Pericolonic tumor deposits in patients with T3N+MO colon adenocarcinomas: markers of reduced disease free survival and intra- abdominal metastases and their implications for TNM classification. Cancer. 2000; 88:2228-2238.

24. **Turner JR**, Liu L, Fligiel SE, Jaszewski R, and Majumdar AP. Aging alters gastric mucosal responses to epidermal growth factor and transforming growth factor-alpha. Am J Physiol Gastrointest Liver Physiol. 2000; 278:G805-810.

25. **Turner JR**, Black ED, Ward J, Tse CM, Uchwat FA, Alli HA, Donowitz M, Madara JL, and Angle JM. Transepithelial resistance can be regulated by the intestinal brush border Na+-H+ exchanger NHE3. Am J Physiol Cell Physiol. 2000; 279:C1918-1924.

26. **Turner JR**, Cohen DE, Mrsny RJ, and Madara JL. Noninvasive in vivo analysis of human small intestinal paracellular absorption: regulation by Na+-glucose cotransport. Digestive diseases and sciences. 2000; 45:2122-2126.

27. **Turner JR**, Torres CM, Wang HH, Shahsafaei A, Richards WG, Sugarbaker D, and Odze RD. Preoperative chemoradiotherapy alters the expression and prognostic significance of adhesion molecules in Barrett's-associated adenocarcinoma. Hum Pathol. 2000; 31:347-353.

28. Yoshimura FK, Wang T, Yu F, Kim HR, and **Turner JR**. Mink cell focus-forming murine leukemia virus infection induces apoptosis of thymic lymphocytes. J Virol. 2000; 74:8119-8126.

29. Berglund JJ, Riegler M, Zolotarevsky Y, Wenzl E, and **Turner JR**. Regulation of human jejunal transmucosal resistance and MLC phosphorylation by Na+-glucose cotransport. Am J Physiol Gastrointest Liver Physiol. 2001; 281:G1487-1493.

30. Furuta GT, **Turner JR**, Taylor CT, Hershberg RM, Comerford K, Narravula S, Podolsky DK, and Colgan SP. Hypoxia-inducible factor 1-dependent induction of intestinal trefoil factor protects barrier function during hypoxia. J Exp Med. 2001; 193:1027-1034.

31. Guzman CB, Walsh M, Reddy V, Donthireddy V, Mahmood F, Bode A, **Turner JR**, Jacober SJ, and Sowers JR. Altered myosin light-chain phosphorylation in resting platelets from premenopausal women with diabetes. Metabolism. 2001; 50:151-156.

32. Kinzie JL, Naylor PH, Nathani MG, Peleman RR, Ehrinpreis MN, Lybik M, **Turner JR**, Janisse JJ, Massanari M, and Mutchnick MG. African Americans with genotype 1 treated with interferon for chronic hepatitis C have a lower end of treatment response than Caucasians. J Viral Hepat. 2001; 8:264-269.

33. Nusrat A, von Eichel-Streiber C, **Turner JR**, Verkade P, Madara JL, and Parkos CA. Clostridium difficile toxins disrupt epithelial barrier function by altering membrane microdomain localization of tight junction proteins. Infect Immun. 2001; 69:1329-1336.

34. **Turner JR** and Black ED. NHE3-dependent cytoplasmic alkalinization is triggered by Na(+)-glucose cotransport in intestinal epithelia. Am J Physiol Cell Physiol. 2001; 281:C1533-1541.

35. Yu Y, Rishi AK, **Turner JR**, Liu D, Black ED, Moshier JA, and Majumdar AP. Cloning of a novel EGFR-related peptide: a putative negative regulator of EGFR. Am J Physiol Cell Physiol. 2001; 280:C1083-1089.

36. Abner SR, Hill DE, **Turner JR**, Black ED, Bartlett P, Urban JF, and Mansfield LS. Response of intestinal epithelial cells to Trichuris suis excretory-secretory products and the influence on Campylobacter jejuni invasion under in vitro conditions. J Parasitol. 2002; 88:738-745.

37. Edens HA, Levi BP, Jaye DL, Walsh S, Reaves TA, **Turner JR**, Nusrat A, and Parkos CA. Neutrophil transepithelial migration: evidence for sequential, contact-dependent signaling events and enhanced paracellular permeability independent of transjunctional migration. J Immunol. 2002; 169:476-486.

38. Kles KA, **Turner JR**, and Tappenden KA. Enteral nutrients alter enterocyte function within an in vitro model similar to an acute in vivo rat model during hypoxia. J Parenter Enteral Nutr. 2002; 26:71-76.

39. Zolotarevsky Y, Hecht G, Koutsouris A, Gonzalez DE, Quan C, Tom J, Mrsny RJ, and **Turner JR**. A membrane-permeant peptide that inhibits MLC kinase restores barrier function in in vitro models of intestinal disease. Gastroenterology. 2002; 123:163-172.

40. Clayburgh DR, Rosen S, Witkowski ED, Wang F, Blair S, Dudek S, Garcia JG, Alverdy JC, and **Turner JR**. A differentiation-dependent splice variant of myosin light chain kinase, MLCK1, regulates epithelial tight junction permeability. J Biol Chem. 2004; 279:55506-55513.

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42. Wang J, Anders RA, Wu Q, Peng D, Cho JH, Sun Y, Karaliukas R, Kang HS, **Turner JR**, and Fu YX. Dysregulated LIGHT expression on T cells mediates intestinal inflammation and contributes to IgA nephropathy. J Clin Invest. 2004; 113:826-835.

43. Wu L, Zaborina O, Zaborin A, Chang EB, Musch M, Holbrook C, Shapiro J, **Turner JR**, Wu G, Lee KY, and Alverdy JC. High-molecular-weight polyethylene glycol prevents lethal sepsis due to intestinal Pseudomonas aeruginosa. Gastroenterology. 2004; 126:488-498.

44. Zhao H, Shiue H, Palkon S, Wang Y, Cullinan P, Burkhardt JK, Musch MW, Chang EB, and **Turner JR**. Ezrin regulates NHE3 translocation and activation after Na+-glucose cotransport. Proc Natl Acad Sci U S A. 2004; 101:9485-9490.

45. Arvans DL, Vavricka SR, Ren H, Musch MW, Kang L, Rocha FG, Lucioni A, **Turner JR**, Alverdy J, and Chang EB. Luminal bacterial flora determines physiological expression of intestinal epithelial cytoprotective heat shock proteins 25 and 72. Am J Physiol Gastrointest Liver Physiol. 2005; 288:G696-704.

46. Clayburgh DR, Barrett TA, Tang Y, Meddings JB, Van Eldik LJ, Watterson DM, Clarke LL, Mrsny RJ, and **Turner JR**. Epithelial myosin light chain kinase-dependent barrier dysfunction mediates T cell activation-induced diarrhea in vivo. J Clin Invest. 2005; 115:2702-2715.

47. Gill RK, Saksena S, Tyagi S, Alrefai WA, Malakooti J, Sarwar Z, **Turner JR**, Ramaswamy K, and Dudeja PK. Serotonin inhibits Na+/H+ exchange activity via 5-HT4 receptors and activation of PKC alpha in human intestinal epithelial cells. Gastroenterology. 2005; 128:962-974.

48. Kimura Y, **Turner JR**, Braasch DA, and Buddington RK. Lumenal adenosine and AMP rapidly increase glucose transport by intact small intestine. Am J Physiol Gastrointest Liver Physiol. 2005; 289:G1007-1014.

49. Kles KA, Vavricka SR, **Turner JR**, Musch MW, Hanauer SB, and Chang EB. Comparative analysis of the in vitro prosecretory effects of Balsalazide, Sulfasalazine, Olsalazine, and Mesalamine in Rabbit Distal Ileum. Inflamm Bowel Dis. 2005; 11:253-257.

50. Kohler JE, Zaborina O, Wu L, Wang Y, Bethel C, Chen Y, Shapiro J, **Turner JR**‡, and Alverdy JC‡. Components of intestinal epithelial hypoxia activate the virulence circuitry of Pseudomonas. Am J Physiol Gastrointest Liver Physiol. 2005; 288:G1048-1054.

51. Marski M, Kandula S, **Turner JR**, and Abraham C. CD18 is required for optimal development and function of CD4+CD25+ T regulatory cells. J Immunol. 2005; 175:7889-7897.

52. Owens SE, Graham WV, Siccardi D, **Turner JR**, and Mrsny RJ. A strategy to identify stable membrane-permeant peptide inhibitors of myosin light chain kinase. Pharm Res. 2005; 22:703-709.

53. Russo JM, Florian P, Shen L, Graham WV, Tretiakova MS, Gitter AH, Mrsny RJ, and **Turner JR**. Distinct temporal-spatial roles for rho kinase and myosin light chain kinase in epithelial purse-string wound closure. Gastroenterology. 2005; 128:987-1001.

54. Savkovic SD, Villanueva J, **Turner JR**, Matkowskyj KA, and Hecht G. Mouse model of enteropathogenic Escherichia coli infection. Infect Immun. 2005; 73:1161-1170.

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60. Wang F, Graham WV, Wang Y, Witkowski ED, Schwarz BT, and **Turner JR**. Interferon-gamma and tumor necrosis factor-alpha synergize to induce intestinal epithelial barrier dysfunction by up-regulating myosin light chain kinase expression. Am J Pathol. 2005; 166:409-419.

61. Wang J, Anders RA, Wang Y, **Turner JR**, Abraham C, Pfeffer K, and Fu YX. The critical role of LIGHT in promoting intestinal inflammation and Crohn's Disease. J Immunol. 2005; 174:8173-8182.

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Narrative Report

I am an investigator and gastrointestinal surgical pathologist with administrative responsibilities that have included service as Associate Chair, editor and editorial board member, and study section chair and member. My annual effort has focused on research with significant clinical service, administrative, teaching, and national leadership. My major laboratory accomplishments include discovery of fundamental and translational aspects of epithelial transport and barrier regulation in response to physiological and pathophysiological stimuli.

My laboratory focuses on epithelial and mucosal biology using the gastrointestinal tract and related diseases as organotypic models. In early work we discovered the essential role of myosin light chain kinase (MLCK) in physiological tight junction regulation, identified subsequent activation of NHE3-mediated Na^+ absorption, and defined the mechanisms responsible for this coordinated paracellular and transcellular transport.

My group then asked if these regulatory mechanisms were relevant to disease pathogenesis. We found that MLCK triggers endocytosis of the tight junction protein occludin to effect barrier loss in acute, tumor necrosis factor α- (TNF-) induced diarrhea and discovered that TNF-induced diarrhea required both barrier loss and NHE3 downregulation. We cloned and characterized intestinal epithelial MLCK, its promoter, alternative splicing, and transcriptional upregulation by TNF in experimental and human

inflammatory bowel disease (IBD). We have created and used genetically-modified mice to define the critical contributions of intestinal epithelial MLCK to intestinal and systemic disease, including IBD and graft versus host disease.

Our work has defined distinct high capacity-high selectivity (pore) and low capacity-low selectivity (leak) tight junction flux pathways and their regulation by pathogenic stimuli. MLCK regulates the leak pathway, while claudin-2 mediates pore pathway conductance. Recently, we performed single channel conductance analyses of trans-tight junction, claudin-2 channels and showed them to be highly dynamic. We are now characterizing relative contributions and integration of pore and leak pathway flux in infectious and immune-mediated diseases.

My laboratory has led efforts to image tight junction regulation in vitro and in vivo. We have used these technologies to discover continuous molecular remodeling of tight junction structure and protein interactions at steady-state, mechanisms of homeostatic and pathophysiological tight junction regulation, and specific contributions of tight junction proteins to intraepithelial lymphocyte migration and innate defense.

The research above has served as a training vehicle for high school and graduate students as well as postdoctoral fellows, many of whom now hold faculty positions. Nearly all have had one or more first author papers, won awards, and been awarded grants and fellowships.

My clinical, educational, and administrative activities are detailed elsewhere. Over time, my extensive classroom efforts have been superseded by authorship of chapters in major textbooks of pathology, surgical pathology, gastroenterology, and physiology. Finally, I have taken on major responsibilities as editor and editorial board member of leading journals and in multiple professional societies.

In summary, I am a physician scientist who has made and continues to make contributions to our understanding of epithelial and mucosal biology and gastrointestinal pathophysiology while contributing actively to education, mentorship, clinical practice, administration, and society leadership.

Exhibit D

regarding olmesartan?

A. No.

Q. The first time that you spoke with Mr. Babington was March 31, 2016, according to Exhibit 6, correct?

A. We may have had e-mails before that, but something along those lines, yes.

Q. It says there was a document review. What documents did you review at that initial meeting, do you know?

A. I think they sent me a few articles related to olmesartan.

Q. Do you remember what articles they were?

A. I couldn't tell you exactly. I would -- I'd be speculating. I could speculate.

Q. Have you published any articles with regard to olmesartan?

A. No, I have not.

Q. Have you given any presentations regarding olmesartan?

A. I have not.

Q. Am I correct that before you were retained in this litigation, you had no interest

in olmesartan?

A. Other than the medical literature that reported it.

Q. Meaning you were familiar with the fact that some articles had been published in the literature, but other than that awareness you had no interest in olmesartan, correct?

A. That's right.

Q. You know Joseph Murray, correct?

A. Yes, I do.

Q. You respect him?

A. Yes.

Q. Is he considered perhaps one of the most -- rephrase.

Is he considered the or one of the most respected celiac specialists in the world?

A. I think he's one of the most respected, absolutely.

Q. Joseph Murray is the world's authority regarding celiac and other disease processes, correct?

A. I think specifically regarding celiac disease, yes.

Q. Have you ever spoken with Dr. Murray

regarding olmesartan?

A. I have not.

Q. Have you ever attended a presentation regarding olmesartan?

A. I have not.

Q. Before you were contacted to act as an expert in this litigation, what, if any, articles regarding olmesartan were you familiar with specifically?

A. You know, I've been looking at a lot of articles in the months since then, and I don't think I could give you a clear list of what I had seen before and what I hadn't. I'm sure I'd seen the 2012 Mayo Clinic report. And I'm sure I'd seen some other papers, but I couldn't specifically tell you which ones.

Q. The second invoice of May 24 bills for document preparation, protocol, phone call preparation, and phone call with Mr. Babington on May 14.

Do you see that?

A. Yes.

Q. It says you spent two and a half hours on that day?

A. No. The phone call was on May 14. I think the two and a half hours total was between April 23rd and May 24th.

Q. Okay. Let me ask you again.

The two and a half hours that you billed would have encompassed all of those activities you described between April 23 and the date of this invoice, May 24?

A. Yes.

Q. What protocol are you referring to?

A. Mr. Babington asked me to put together a rough protocol of, if we were going to approach these biopsies in a standardized way, how we might go about doing that.

Q. These were biopsies of patients who were having their cases reviewed as part of the litigation?

A. Yes.

Q. In your clinical practice, it's my understanding that you spend most of your time on research related activities, is that correct?

A. Yes, about 70 percent.

Q. You're at Brigham & Women's now. When did you return to Brigham & Women's?

A. Last February.

Q. At Brigham & Women's, do you have any clinical responsibilities?

A. Yes.

Q. What are your clinical responsibilities at the Brigham?

A. It's exclusively GI pathology, almost exclusively biopsies.

Q. How many hours a week do you have clinical responsibility at Brigham & Women's?

A. I have eight weeks per year, so you can break that out any way you like.

Q. Since you went to Brigham & Women's, how many times have you looked at biopsies in connection with suspected or identified celiac disease?

A. I couldn't give you an exact number, but I'm sure it's quite a few. We see a lot of those.

Q. Estimate for me, how many have you seen?

A. Well, an estimate, maybe a couple hundred.

Q. In the time you've been at Brigham &

which did have the clinical picture of olmesartan enteropathy that aren't listed? Do you know whether there are any such reports?

A. So you've made a conclusion in your question that I can't agree with.

Q. Do you know whether there are other reports that aren't listed in that report where patients either had diarrhea and weight loss or serious diarrhea that aren't included?

A. I don't know that.

Q. Do you know who Crawford Parker is?

A. No, I don't, other than his title here.

Q. With regard to the Caspard report, what, if anything, about it is significant to you?

A. You know, I can't remember anything specific. I'm sorry that I don't have a copy of it with me.

Q. With regard to the less than five MedWatch reports that you reviewed a week or so ago, or in the last week, what, if anything, can you specifically point to in your deposition right now that you would say is significant to

you about those MedWatch reports, specific to each one?

A. I think that what's specific to the MedWatch reports is the same thing that I found in each of the case reports, is that they were wholly uncontrolled, and showed correlation, but no data really supporting causation.

Q. What does correlation mean?

A. Correlation means if something, A, happened, then more often than not B will follow or be accompanying that.

Q. The MedWatch reports you saw you believe did show a correlation between olmesartan and gastrointestinal illness, correct?

A. In some individual cases there was a correlation.

Q. Were you shown any adverse event reports in which Daiichi performed a causality assessment internally, when their physicians actually evaluated causality?

A. I don't think so.

Q. Do you know whether Daiichi actually, their internal physicians actually evaluated any

peer-reviewed medical literature you can point to that concludes explicitly that there is no association between olmesartan and what has been termed in the literature olmesartan-associated enteropathy or sprue-like enteropathy? Any article that reaches that conclusion?

A. Yes, there's several.

Q. That says there's no association?

A. That says there was no association detected in their study.

Q. Which articles say there's no association. Well, let me stop you for a second, stop you for a second.

My question is not somebody saying whether or not they found an association in their study. My question is, is there any article in the peer-reviewed literature where there is an explicit conclusion that olmesartan is not associated with olmesartan-associated enteropathy as that term is used in the literature, or sprue-like enteropathy, any article that reaches that explicit conclusion?

A. Well, I think failure to detect an association in a highly powered study would come

to that conclusion to the extent that it's possible to prove a negative. Proving a negative is obviously impossible or very difficult. So it depends how you interpret that.

MR. SLATER: Move to strike.

Q. Is there any article that reaches that conclusion that I just asked you, explicitly states there is no association?

A. Yes.

Q. Are you saying that there's articles where a study was done and they did not find an association in their study? Because that's not what I'm asking you.

MR. PARKER: Objection.

BY MR. SLATER:

Q. That's a different conclusion than what I'm asking you about.

MR. PARKER: Objection. Argumentative.

You may answer.

A. I'm saying --

BY MR. SLATER:

Q. Do you understand my question?

A. Associated with, you would like to see some evidence of a controlled rechallenge that really told you that it was due to olmesartan, or at least a highly controlled dechallenge in which you could say it was associated. Obviously a dechallenge would never be sufficient in a single case for making that conclusion of causation, but it probably could be sufficient for saying in that patient it's associated and maybe we should just sort of not worry about it and give that patient some other drug.

Q. There are some highly controlled dechallenges in the case reports published in the literature, right?

A. I don't believe so.

Q. Okay. If you have a patient who -- let me rephrase.

I'm going to give you a hypothetical patient and ask you a question about the patient. Okay?

A. Okay.

Q. If you have a patient who is taking olmesartan for more than two years, and more

documents to describe this condition that we're talking about?

A. I don't believe I saw it in the documents that I saw.

Q. Would it be helpful to you to know that -- rephrase.

If, in fact, Daiichi in its own internal documents refers to the condition as olmesartan induced enteropathy, that would be significant to you, since they presumably are experts regarding the side effects caused by their drug, right?

MR. PARKER: Objection.

A. It depends. It would certainly be something to be aware of, but it wouldn't be the only factor in my thinking, because then I would be just accepting their conclusion based on data I haven't seen. So it would really depend on why they were making that conclusion.

BY MR. SLATER:

Q. If there are physicians employed by Daiichi who found in reviewing adverse event reports that gastrointestinal illness representing sprue-like enteropathy in terms of

BY MR. SLATER:

Q. Did you find it, Doctor?

A. No, I'm not finding an actual reprint of the label, I'm sorry.

Q. You have the FDA safety notification right in front of you, right?

A. Yes, I do.

Q. Right in the beginning it says that "Olmesartan can cause sprue-like enteropathy," right? Doctor, the very first paragraph, do you see it?

A. Yes. And I think they're using the regulatory phraseology, so they're saying "can" as in it's possible that they caused these intestinal problems.

Q. Doctor, it says right on the drug safety communication, July 3, 2013, "The US Food and Drug Administration (FDA) is warning that the blood pressure drug olmesartan medoxomil (marketed as Benicar, Benicar HCT, Azor, Tribenzor, and generics) can cause intestinal problems known as sprue-like enteropathy." That's the words, right?

A. That's the words.

Q. Do you disagree with the FDA?

A. No.

Q. Now, going to Exhibit 8, Brigham & Women's Hospital is telling people who want to read their health library that side effects that patients can suffer as a result of taking olmesartan include diarrhea, vomiting, and weight loss, that's what your institution is telling patients, correct?

A. Yes.

Q. And that's truthful -- let me rephrase.

And that is truthful information, correct?

A. They're saying what side effects may I notice from receiving this medication. So they're listing that as a possible side effect that you should report to your doctor or health care professional.

Q. The reason Brigham & Women's provides possible side effects here in that library is so patients will be aware of side effects that your institution thinks can occur in some patients due to taking the drug, right?

entity?

A. I don't think it's been definitively shown to be true, to be an entity.

Q. If the people -- I'd like you to assume -- a hypothetical. I'm going to ask you a new question. I'd like you to assume that the people who believe olmesartan-associated enteropathy is a real clinical entity are correct and that you're wrong, I'd like you to assume that, okay?

A. Okay.

Q. Assuming that to be true, there is evidence that it has a patchy appearance on biopsy, correct?

A. I believe that's what's been reported.

Q. Looking at Exhibit 9, this letter from Drs. Gallivan and Brown, in the last paragraph they state in the second sentence, "Thus, it is important to consider olmesartan induced enteropathy in patients with histological sprue-like findings, with or without colonic inflammation, in the absence of other celiac disease or other medical condition."

Do you see that?

A. Yes.

Q. You agree with that statement in terms of that clinicians should consider olmesartan induced enteropathy under those circumstances, correct?

A. For the reasons we discussed, I wouldn't agree with the phraseology. I would agree that you'd want to consider taking your patient off olmesartan if they failed a gluten-free diet and have appropriate histopathology, and so on.

Q. The reason that a clinician would take a patient off of olmesartan in the setting of the clinical features of sprue-like enteropathy is because the clinician thinks that the olmesartan may be causing the clinical syndrome, correct?

A. They recognize it as a possibility, yes.

Q. And where the only change that's made is the withdrawal of the olmesartan in a particular patient, and the patient's clinical syndrome resolves, the symptoms go away, the pathology normalizes, in that case, all other

things being equal, olmesartan-associated enteropathy should be on the differential diagnosis as the cause of that clinical presentation, correct?

MR. PARKER: Objection.

A. I think permanent withdrawal of olmesartan in their drug regimen would be a reasonable practice.

BY MR. SLATER:

Q. And the reason why it would be reasonable to permanently withdraw the olmesartan is because, and we'll start small here, of the possibility that the olmesartan was causing the clinical syndrome, correct?

A. Sure, that remains a possibility.

Q. Where the only change made is the withdrawal of the olmesartan, and the patient then has resolution of the clinical symptoms and the pathology normalizes, in the absence of any other change for that patient, the olmesartan is the likely cause of the clinical syndrome that was being suffered by patient, correct?

MR. PARKER: Objection.

BY MR. SLATER:

Q. From a clinical perspective for that patient, correct?

A. That would indicate a correlation, and a good management decision in the patient. It does not indicate that olmesartan caused that.

Q. It indicates that olmesartan, from a clinical perspective, was the likely cause of the clinical symptoms that ceased and normalized and got all better when the patient stopped taking the olmesartan if it was the only change that the patient had, correct?

A. Again, I --

Q. Clinically.

A. I think clinically it tells you that it would be a good idea to change the medication regimen for that patient. You can theoretically say maybe it was the cause, let's not give this patient olmesartan. But I don't think you can conclude that olmesartan was the cause.

Q. Is it your testimony the only way that you can conclude the olmesartan was the cause if you then were to put the patient back on the olmesartan, and the symptoms and the pathology were to recur?

A. You know, you really need -- in these sorts of cases we know the placebo effects can be strong, you really need controlled tests. That's really the only way to do it. Really case reports are the weakest form of data in the medical literature, that's well-recognized throughout the medical literature, you really can't draw these conclusions from uncontrolled case reports like this.

Q. So you're saying that you'd need to see a controlled rechallenge to prove causation in that case, right?

MR. PARKER: Hold on. Technical problem.

A. I'm saying we need a controlled and properly done randomized rechallenge, and then you can make a determination about one patient.

BY MR. SLATER:

Q. If it causes it in one patient, then that would answer the question that there is general causation, correct?

MR. PARKER: Objection.

A. If it causes it in one patient by some idiosyncratic reaction where it unmasks a

Q. Dr. Murray is one of the world's authorities on this issue, correct?

A. On celiac disease, absolutely. Is that what you're asking?

Q. And do you know -- and you've actually co-authored a publication with Dr. Murray, is that right?

A. I think we're co-authors of a publication, might be two.

Q. The second sentence in the abstract -- I'm sorry, did I interrupt you?

A. Yeah. I was going to say there might be more than one that I'm co-authors with Joe on, but I'm not certain.

Q. Okay. Have you ever told him that you think he publishes in garbage medical journals?

A. You know, I think probably everybody has published papers in garbage medical journals from time to time. And I think if I told him maybe not in such crude terms that I thought this was not really the best journal, I think he'd agree.

Q. Are very good articles with very good science published in garbage medical journals

A. In clinical terms.

BY MR. SLATER:

Q. If the physician then says to the patient, okay, you seem like you're better, let's put you back on the olmesartan, and the patient has the clinical symptoms recur, you would agree at that point it's very reasonable for the physician to say, I'm going to pull you back off the olmesartan and we're going to find a different medication for your high blood pressure?

A. Sure.

Q. That's a reasonable clinical decision, right?

A. That's a completely reasonable clinical decision.

Q. And if the physician were to make that decision based upon their clinical judgment that the olmesartan was causing the condition, that would be a reasonable clinical judgment based on those facts, correct?

A. I think you have to ask what you're implying there. If you're implying causation in the scientific or legal sense, no, they don't

have sufficient data to say that. If you're implying in a very loose sense that it might cause it in this patient, it seems like whenever this patient is off olmesartan they're better, we should just use something else because there's not much harm to that, then they can think about it in any terms they like.

MR. SLATER: Well, move to strike.

Q. My question is very specific. If the doctor told the patient, you got better when we took you off the olmesartan, you got sick again when we put you back on the olmesartan, I think that the olmesartan is causing your condition so you should use a different hypertension drug, and you shouldn't take the olmesartan anymore, based on that clinical picture in that clinical context, that's a reasonable medical judgment by that physician, correct?

MR. PARKER: Objection. Asked and answered.

A. I think with some paraphrasing that's correct. I think it might be more appropriate to say I think there's a chance that it's causing that, let's put you on something else

That's all that's here is case reports.

Q. There's not only isolated case reports, are there, Doctor?

A. There's just case reports and small series of 10, 20 patients.

Q. What would you like to do to prove this? Would you like to see somebody actually structure a randomized controlled trial to study this question?

A. You could do that. You could do animal studies. You could do case control studies. You could do controlled dechallenge/rechallenge. There's a lot of things you could do. None of it has been done -- well, some of it has been done. To the extent it's been done, none of it has shown a clear causation.

Q. Okay. Let's start with RCTs.

Who is going to -- how many patients would you need to put into an RCT to study whether or not patients get sprue-like enteropathy from the use of olmesartan? You have no idea how many patients you'd need, right?

Q. I thought you said you would include.

A. No, I said I would exclude pretty much everything we've talked about, if we're talking about sufficient statistical power, and I would consult an epidemiologist about those three studies that I mentioned, because I'm not in a position to analyze the math and see -- ad be able to state definitively were they sufficiently powered. But what I would conclude is that pretty much everything else that we've talked about is case reports, and is not sufficiently powered.

You asked me earlier if the case reports all go on the side of olmesartan does cause enteropathy, and I said yes. But if we're going to use that low bar of how we pile things up, then the Greywoode study certainly must go on the other side. It's at least as good as the case reports.

Q. Do you have the Lagana study handy, the abdominal pain?

A. Yes.

MR. TURNER: I'm sorry. I missed that. What study?

this question.

There are a number of rechallenges documented in the peer-reviewed literature, correct?

A. Uncontrolled rechallenges, yes.

Q. They're not just one, there's a number?

A. Yes.

Q. Correct?

A. Yes.

Q. Taken together, you must agree that there is significance to the number of rechallenges documented, even if they're uncontrolled, there is some significance to that, and it must weigh in the analysis, correct?

A. You need to know what you're pulling from. If you're cherry-picking just the cases where rechallenge was positive, then you can't conclude that. If you tell me that that represents 10 percent of the rechallenges and 90 percent rechallenge didn't do anything, then you would immediately drop that question and conclude that it was a ridiculous question.

So we just don't have the information to assess that, and that's part of the reason these case reports are not useful.

Q. The reports of rechallenges are numerous enough where the rechallenge resulted in resumption of symptoms, there are enough that you have to at least factor them into the analysis of general causation, correct? They have to be part of the analysis, correct?

A. They should be considered, absolutely. Everything that you can find, all data that are available should be considered, and these would be under that umbrella.

Q. The same would hold true for the dechallenges that were positive that showed the people getting better, that's also part of the data that should be analyzed in this question on general causation, correct?

A. Absolutely.

Q. Ultimately in forming an opinion on this, you can only go with the data that's available to you, correct?

A. Correct.

Q. And on a smaller scale, if you take a

the reuse of olmesartan as described? Is that your opinion?

A. My opinion is that some of these could be idiosyncratic drug reactions, some of these could be the result of something else they didn't pick up in their analyses, but that in none of these cases is there proof that olmesartan causes the enteropathy.

Q. You would agree with me that with regard to the patients who had the positive dechallenges and the positive rechallenges as discussed here, that it's possible that for at least some of those patients olmesartan was causing their clinical picture? You'll agree with that, correct?

A. It is possible. I would agree with that.

Q. You just would want to see more rigorous data in order to be willing for you to say I think it's likely, do I understand?

A. I think if you want to prove causation, you need stronger data than this, yes. I think anybody would agree with that.

MR. SLATER: Do we have that Kulai

an opinion as to what was causing it, as you sit here right now?

A. There's a range of things that could have been investigated in these patients. I can't tell you specifically in any case because, again, there's not sufficient data.

Q. Now, looking at your report, Page 5, at the very bottom, you state, "Although some of these studies have merit, it is also reasonable to conclude that Rubio-Tapia's small series stimulated investigators who were eager to join the phenomenon."

That's what you wrote, right?

A. Right.

Q. First of all, which of the studies have merit?

A. I think the studies we've been talking about have merit. The question is whether they prove causation. We disagreed on whether they proved causation. I don't think they're completely useless studies, they are of interest, they do bring people's attention to things.

Q. Now, are there any investigators you

person's weekend.

A. I couldn't put a number on it. I'd say at least 15 years, perhaps more. I'm sure it's more.

Q. With regard to NSAIDs, how long has that been known?

A. Much more than that.

Q. How about with mycophenolate?

A. I think that's a newer drug, so I think it's less, but I would probably say at least ten years.

Q. For clinical physicians who are actually treating patients who have the clinical syndrome that's been identified in the literature as olmesartan enteropathy, in order to treat their patients, do they need any more studies than what's out there, or is there sufficient information for them to know what this entity is as described, and to use that information to treat their patients?

A. So first, I think it's been referred to mostly as olmesartan-associated enteropathy, not olmesartan enteropathy.

Second, I think they have enough

information to be aware of it as a possible entity, and if their patient -- and to do a therapeutic trial by withdrawing the medication. If their patient does well, then they shouldn't put the patient back on olmesartan because there are plenty of alternatives, and they don't need more information for patient management.

Q. So the state of the scientific literature is sufficient to provide the physicians who actually have to treat patients in this area with the information they need to treat the patients, fair statement?

A. Fair statement.

Q. Okay. Doctor, unless Mr. Parker reminds me of things I forgot to ask you, or asks any really, really insightful questions, I will probably not ask you more questions. But if he does, I will probably follow up. So his turn.

THE VIDEOGRAPHER: If we could just go off the record for a moment, please.

Going off the record. The time is 4:42.

(Whereupon, a recess was taken.)

villous atrophy cases, that's one of the top three diagnoses.

Q. So -- never mind. Let me rephrase and go on.

Doctor, you were asked by Mr. Slater about whether rechallenge was, I think his word was strong evidence of causation. Do you recall that series of questioning early this morning?

A. Yes.

Q. And in response to a number of his hypotheticals, you responded that it was an uncontrolled rechallenge.

Do you recall that?

A. Yes.

MR. SLATER: Objection.

BY MR. PARKER:

Q. Can you share with the jury what, if any, importance there is on the question of causation if a rechallenge is controlled versus uncontrolled?

A. Sure. A rechallenge essentially involves taking a patient who has recovered from whatever their illness is, in this case it's one of these patients who seems to do better after

stopping olmesartan, and then giving them olmesartan and asking whether they manifest the disease again, and I'll put that in quotes.

The issues with doing it just in that way, which is more or less the way it's been done, except usually the readministration has not been intentional, the issue with doing it just in that way is that you don't know what else is going on, you haven't controlled for other variables, which there may be many. Most of these patients have been identified when they're reasonably ill.

The second issue, and I think this is really a big one, is the placebo effect. So we know that in trials, in clinical trials, patients receiving placebo often improve. And so if you really want to ask is this a cause or effect, so in this case we're talking about rechallenge, you don't want to ask if I give this patient placebo does their disease recur, and to my knowledge, that hasn't been done. And I think even to prove causality in an individual case, you need data from that patient showing that you've done a randomized trial.

Q. Doctor, for the better part of 20 years before the auto antibodies were identified that are specific to celiac disease, was rechallenge the standard of care for diagnosing that condition?

A. Yes, it was.

MR. SLATER: Objection.

BY MR. PARKER:

Q. Doctor, is there anything that you've seen in the literature regarding the severity of the symptoms in what's reported to be olmesartan-associated enteropathy that would suggest that if you really did want to find cause that you couldn't do a rechallenge in the way that you've described?

A. You certainly wouldn't do it at the trough of their disease when they're at their sickest. But if the reports that say they recover completely, gain back their weight, and don't have malabsorption anymore are true, I don't see any reason why a short-term rechallenge in the manner that I just described, a randomized controlled trial, I don't see any reason that couldn't be done.

Q. And for many years, what did the standard of care require in terms of rechallenge for diagnosing celiac disease?

A. For diagnosis of celiac disease, it required rechallenge with gluten. I think that's really the key difference here, if I can opine for a second.

Q. Sure.

A. So in the case of celiac disease, especially at that time in history, a gluten-free diet was a huge difficulty. It's so much easier today because it's become a popular thing, and with the increasing presence of celiac disease and gluten-sensitive patients who don't have celiac disease, gluten-free diets are everywhere, gluten-free foods are everywhere. It's much easier. In those days, it was really, really hard to do a gluten-free diet, so you wanted to be sure.

In the case here of olmesartan, the treatment is really easy. So if you're wrong and it wasn't olmesartan, it really didn't hurt anybody. There's no cost. And I think that's why if you're managing an individual patient,

Exhibit E

2016 WL 6652358
Only the Westlaw citation is currently available.
United States District Court,
D. New Jersey,
Camden Vicinage.

In re: Benicar (Olmesartan)
Products Liability Litigation.

Master Docket No. 15-2606 (RBK/JS)
|
Signed 11/08/2016
|
Filed 11/09/2016

OPINION

KUGLER, United States District Judge

***1** Plaintiffs' submitted a request (Doc. No. 925) for leave to file a motion for partial summary judgement on the issue of general causation and accompanied it with fourteen (14) exhibits, either excerpts from defendants' depositions or documents produced by defendants. Plaintiffs assert the exhibits are defendants' admissions of general causation, which show that defendants' pharmaceuticals caused plaintiffs' complained of sprue-like enteropathy ["SLE"]. This opinion accompanies the order denying plaintiffs' request without prejudice (Doc. No. 938) and sets forth the reasons therefor.

I. Fact and Procedural Background

This Multidistrict Litigation ("MDL") involves approximately 1900 plaintiffs, who ingested defendants' olmesartan-containing prescription drugs [1] to alleviate hypertension. The named defendants are Daiichi Sankyo, Inc., Daiichi Sankyo Co., Ltd., Daiichi Sankyo U.S. Holdings, Inc., Forest Laboratories, LLC, Forest Laboratories, Inc., Forest Pharmaceuticals, Inc., and Forest Research Institute, Inc. The Daiichi defendants designed, manufactured and sold the drugs at issue. [2] For a time the Forest defendants marketed the drugs. Daiichi Sankyo, Inc. and Daiichi Sankyo U.S. Holdings, Inc. are U.S. companies. Daiichi Sankyo, Inc. is a wholly-owned subsidiary of Daiichi Sankyo U.S. Holdings, Inc. which operates as a holding company. Daiichi Sankyo Co., Ltd. is the parent company of Daiichi Sankyo U.S. Holdings, Inc. Daiichi Sankyo, Inc. operates as the commercial home office and U.S. corporate headquarters of Daiichi Sankyo Co., Ltd., which is a Japanese corporation with its principal place of business in Japan. *See generally* Master Answer of Daiichi Defendants ¶¶ 20, 23-27, 30-31 [Doc. No. 82].

In order to put the Plaintiffs' request in context, the court's management plan initially focuses only on general and specific causation issues, that is, whether defendants' drugs caused the complained of SLE symptoms, which include nausea, vomiting, diarrhea and weight loss.

To date, plaintiffs have taken at least twenty (20) depositions of present and former Daiichi U.S. employees and eighteen (18) depositions of present and former Daiichi Japan employees. The first phase of fact discovery regarding causation issues was all but completed by 30 September 2016; [3] the litigation has now entered the next phase with plaintiffs' causation expert reports due 30 November 2016, defendants' expert reports due 31 January 31, 2017, expert depositions to completed by 28 February 2017, and *Daubert* and summary judgment motions due by March 31, 2017. CMO No. 26. [Doc. No. 626]. The date for the *Daubert* hearing has not yet been set. [4]

***2** Turning to plaintiffs' request filed 13 October 2016 [Doc. 925], it comprises a summary of the 14 accompanying exhibits, which are excerpts of defendants' deposition testimony or defendant-produced documents, and characterizes them as admissions that defendants generally caused plaintiffs' injuries. Plaintiffs' request lacks not only an explanation as to how these summaries and excerpts constitute incontestable facts upon which to base a summary judgement motion but also any jurisprudential support that defendant alleged admissions during discovery in and of themselves properly substitute for expert testimony to demonstrate general causation.

Defendants argue that case law requires plaintiffs to offer admissible expert testimony on general causation because, in this case, linking the cause of each plaintiffs SLE injury to defendants' pharmaceuticals is beyond the ordinary understanding of a lay jury. Ds Response at 2. Defendants also argue that the excerpted testimony and documents are insufficient to unequivocally demonstrate that the

pharmaceuticals caused the complained of injury in each of plaintiff's cases. *Id.* at 3.

The issue is whether the deposition excerpts and internal documents proffered by plaintiffs substitute as expert testimony reliable and fit under *Daubert v. Merrill Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993) to sufficiently inform a jury that defendants' pharmaceuticals caused plaintiffs' SLE in these cases.

II. Legal Standard

Courts generally recognize that plaintiffs in products liability cases must offer admissible expert testimony regarding both general causation and specific causation. *See, e.g., In re Mirena IUD Products Liability Litigation*, ___ F. Supp.3d ____ (S.D.N.Y. 2016) 2016 WL 4059224 at *5, citing *Amorgianos v. Nat'l R.R. Passenger Corp.*, 303 F.3d 256, 268 (2d Cir. 2002; *see Rutigliano v. Valley Bus. Forms*, 929 F.Supp. 779, 783 (D.N.J. 1996), *aff'd sub nom. Valley Bus. Forms v. Graphic Fine Color, Inc.*, 119 F.3d 1577 (3d Cir. 1997) and further stating that "substantive law across all relevant jurisdictions holds (reference omitted) that 'where a causal link is beyond the knowledge or expertise of a lay jury, 'expert testimony is required to establish causation' (citations omitted)". *Id.*

Recently, the *Mirena* court found that, although there may be circumstances when defendants' admissions in a product liability case can substitute for expert testimony, those circumstances are "exceedingly rare". *In re Mirena* at *8. Expert testimony is generally required in product liability cases because it prevents the jury from engaging in speculation in determining the causal link between using or ingesting the product and the injuries complained of following that use. *Id.* at *5. Determining that causal link typically requires complex medical information beyond the knowledge, understanding, and experience of a lay juror. Expert testimony typically provides this link. *See generally* Christopher R.J. Pace, *Admitting and Excluding General Causation Expert Testimony: The Eleventh Circuit Construct*, 37 AM. J. TRIAL ADVOC. 47, 51-60 (2013) (comparing the probative value of various general causation methodologies used by experts to support their testimony as *Daubert* reliable).

Purported admissions offered as substitutes for expert testimony must be "clear, unambiguous, and concrete" and suffice to prove general causation without speculation. *Id.* at *8. "They can substitute for expert testimony only when they serve the same purpose as expert testimony, that is, to provide the jury with a scientific, non-speculative basis to assess general causation." *Id.* at *12.

Also recently, a court in the Third Circuit analyzed an analogous issue—whether the plaintiffs were able to establish general causation with virtually no expert testimony, which had been excluded as inadmissible under *Daubert* and Federal Rule of Evidence (FRE) 702. *In re Zoloft Products Liability Litigation*, ___ F. Supp.3d ____ (E.D. Pa. 2016) 2016 WL 1320799. There, plaintiffs found themselves precluded from offering new expert testimony on the issue of general causation—whether Zoloft caused the complained of birth defects—and were left with arguing that other evidence established causation. Such other evidence included declarations by treating physicians of differential diagnoses, case reports by treating physicians of the occurrence of birth defects, defendants' internal documents including literature reviews and published studies relying on statistics about whether Zoloft was the cause of the complained of birth defects, foreign language documents that contained a warning against pregnant women's ingestion of Zoloft, and drafts of product documents. *Id.* at *9.

***3** The *Zoloft* court found that, taken together, plaintiffs' potentially admissible evidence supported only an association between the drug at issue and the complained of birth defect and therefore presented only a possibility of general causation. *Id.* at *10. The court found that "plaintiffs have not produced sufficient admissible evidence from which a reasonable factfinder could determine, by a preponderance of the evidence, that [the drug at issue] could have caused Plaintiff's injuries." *Id.*

Mirena and *Zoloft* resolve the issue raised by plaintiffs' request. Unless information characterized by plaintiffs as defendants' admissions provide to the jury evidence that is clear, unambiguous, and concrete and suffices to prove general causation without the jury's speculation as to complex medical issues, then such information does not substitute for *Daubert*-admissible expert testimony of general causation.

III. Discussion of Proffered Information by Plaintiffs

Each of the 14 exhibits will be reviewed for its sufficiency to substitute as expert testimony that demonstrates general causation without relying on a jury's speculation as to what the exhibit means.

Exhibit 1: 5 page excerpt (out of at least 461 pages) from the deposition of Crawford Parker, MD, defendants' Senior Director of Clinical Safety and Pharmacovigilance ("CSPV") in the United States.
Plaintiffs' request provides no specific reason for including Dr. Parker's testimony, nor contextualizes this excerpt within the deposition as a whole. Dr. Parker's testimony relates to certain documents that Dr. Parker provided to Dr. Peter Green, apparently a medical consultant to defendants, in advance of an unidentified meeting at which SLE adverse events were to be discussed. These documents included: a January 2009 review by defendants of celiac disease AE reports; the defendants knowledge of the 2012 Mayo Clinic publication [5]; the FDA request to Defendants to review SLE adverse events; and a September 2012 review by defendants of SLE adverse events.

Despite plaintiffs' repeated attempts, the excerpt shows that Dr. Parker expressly declined to characterize the information in one of defendants' ROADMAP clinical study as "an analysis". Dr. Parker's testimony does not suffice to inform in a clear, unambiguous, and concrete way and without jury speculation as to the complex medical issues involved in determining the mechanism by which olmesartan may generally cause the complained of injuries. This exhibit does not substitute for *Daubert*-admissible expert testimony of general causation.

Exhibit 2. 1 page excerpt (out of at least 165 pages) from the deposition of defendants' employee in Japan, Akinori Nishiwaki.
Plaintiffs' request states no specific reason for including Nishiwaki san's testimony or contextualizes this excerpt within his deposition as a whole nor was Nishiwaki san's role for defendants identified.

Plaintiffs' attorney read the following sentence from an unidentified document: "Before identifying olmesartan as a cause of villous atrophy, we, too, had considered 30 percent of our seronegative patients to have unclassified sprue". Nishiwaki san was then asked to confirm the presence of the word "cause" in the sentence, which he did. That this one sentence included the word "cause" and that the deponent affirmed the presence of that word is not a clear, unambiguous, concrete, or sufficient demonstration of general causation.

4 *Exhibit 3. 5 page excerpt (out of at least 415 pages) from the deposition of Allen Feldman, MD, head of defendants' CSPV unit in the United States.*
Plaintiffs assert that Dr. Feldman "admitted that the only cause he could identify for the [symptoms] suffered by these patients was Olmesartan [emphasis added]." (Ps Letter Request at 4-5). When asked about the meaning of a statement in a Medwatch report [6] (specifically whether the most likely explanation for patients' symptoms was olmesartan given their history of ingestion and dechallenges and positive rechallenges of the drug), Dr. Feldman replied: "The only cause given here [in the Medwatch report] is olmesartan". Feldman Dep. 285:24.

Dr. Feldman was only asked to confirm what a certain Medwatch report states; he was not asked as a medical professional to admit that olmesartan caused plaintiffs' symptoms. Dr. Feldman's deposition testimony is not clear, unambiguous, concrete or sufficient as to demonstrate general causation.

Exhibit 4: Email of 2 pages dated 26 March 2015, sent jointly from Ford Parker, MD, the same employee as in Exhibit 1, Ulf Stellmacher, director of defendants' CSPV unit in Europe, and Hideki Tagawa, associate *manager of defendants' CSPV unit in Japan, to all employees in each of defendants' CSPV units (in the US, Japan and Europe).*
Having the subject of "Coding and Expectedness of Sprue-Like Enteropathy for Olmesartan and Olmesartan Combination Products", the email states that the code "Syndrome SLE" was added as an expected reaction to the US Product Insert ("USPI") on 3 July 2013 and to defendants' Company Core Data Sheets ("CCDSs") in September 2013. The email provides recommendations to defendants' CSPV on how to determine expectedness [7] because it is believed they may not understand SLE symptoms. To that end, the email identifies "Syndrome SLE" as including "nausea, vomiting and diarrhea, signs typical of olmesartan induced sprue-like enteropathy, such as weight loss." Exhibit 4, at ¶1. The apparent purpose here is to inform CSVP employees how Syndrome

SLE will be reported (presumably by clinicians) and how to code that information in periodic safety reports to regulatory agencies.

***5** The email states: " 'Syndrome sprue-like' is currently the DSPD[8] (the "Daichii Sankyo Pharmaceutical Development" functional unit) recommended term in MedDRA Version 16.1"[9] and advises that a report coded as olmesartan-related intestinal villous atrophy should be handled as a sprue-like enteropathy report, in order to conform with the findings in the Mayo Clinic publication (Rubio-Tapio, *supra*) that first publicly reported that villous atrophy in olmesartan-takers was related to their SLE symptoms. Item 4 of the email recommends that, when a clear diagnosis of SLE is not reported (presumably by a clinician), symptoms of diarrhea, malabsorption, or weight loss should be coded not as SLE, but as separate reactions.

Although stating that there are signs typical of olmesartan induced sprue-like enteropathy, this exhibit expressly informs defendants' employees that these signs, when not accompanied by a clear diagnosis, cannot be reported as SLE. Since the exhibit on its face calls for a clinician's diagnosis before defendants will report olmesartan induced SLE, it cannot provide clear, unambiguous, and concrete proof of general causation. Without more, and in light of the entire email, the mere use by defendants of the term "olmesartan induced sprue-like enteropathy" does not suffice to inform a jury as to general causation.

Exhibit 5: Email from Dr. Ulf Stellmacher to Crawford Parker, MD, and Hideki Tagawa, dated to 16 Jan 2015, attaching a summary of the "3rd fatal SLE case we have just processed". The attachment is a Power Point of 3 slides apparently prepared by Dr. Stellmacher, director of defendant's CSPV unit in Europe.
Slide 2 states that villous atrophy was found in a 70 year-old man living in France who had been taking Olmetec® or Alteis®[10] for an unknown period and who had died. As defendant-manufactured equivalents to the pharmaceuticals issued here, the medications were for hypertension. The slide states that "Causality cannot be denied based on available information. Though diagnosis was not confirmed, this case represents SLE".

This exhibit does not detail the causal link between the injuries complained of and the drugs at issue and cannot demonstrate general causation.

Exhibit 6: 8 page excerpt (out of at least 84 pages) from the deposition of Hideki Tagawa, associate *manager of defendants' CSPV unit in Japan.*
Tagawa san is apparently being asked to comment on the Power Point in Exhibit 5 above. He confirms that it states the cause of the death of a 70 year-old man in France was an olmesartan drug. Tagawa Dep. 58: 9 to 60: 20.

He is then asked to comment on an unidentified Power Point and on other unidentified documents, which leaves this court no point of reference from which to review independently to what Tagawa san is attesting to. Tagawa san confirms that the unidentified Power Point states: (1) based on reports of SLE in the U.S., a causal relationship between olmesartan-containing drugs and severe diarrhea could not be denied; and (2) Japanese and U.S. package inserts were modified to add diarrhea as a serious side effect following the U.S. reports. *Id.* at 69-70. He also confirms that he wrote an email stating that U.S. and Japanese reports indicated that chronic diarrhea improves when olmesartan is stopped in most cases. He states that what he wrote in that email is based on what defendants' medical advisors had told him. *Id. at* 83-84.

***6** Although Tagawa san affirms that he wrote certain content relating to SLE and confirms the statements set forth in the documents before him, he clearly indicates he is not an expert able to independently attest to general causation. This exhibit cannot demonstrate general causation.

Exhibit 7: 3 page excerpt (out of at least 173 pages) from the deposition of Yasushi Hasebe, head of defendants' CSVP unit in Japan.
Plaintiffs' request does not contextualize this excerpt within the deposition as a whole.

Hasebe san was asked: "You're not denying that the olmesartan was one of the factors causing the severe diarrhea, dehydration and hospitalizations described in this adverse report. You're not denying that, right?" Hasebe Dep. 127: 14-19. He answers, "Correct, I think that's one of the factors". *Id.* at 127:21-22.

Inasmuch as Hasebe san's answer would lead to jury speculation as to what other factors caused the complained of injuries, the exhibit is not clear, unambiguous, specific or sufficient to demonstrate general causation.

Exhibit 8: 1 page excerpt (out of at least 152 pages) from the deposition of Mahmoud N. Ghazzi, M.D., Ph.D.
Plaintiffs' request states no specific reason for including Dr. Ghazzi's testimony or contextualizes this excerpt within the deposition as a whole nor was Dr. Ghazzi's role for defendants identified. Research from independent sources identifies Dr. Ghazzi as defendants' Global Head of Drug Development, as well as Head of Daiichi Sankyo Pharmaceutical Department in the U.S.

In response to questions about Deposition Exhibit No. 3068, which was neither provided nor summarized, and which the court, therefore, could not review, Dr. Ghazzi confirmed that, in May 2014, defendants were starting to arrange a meeting with key European opinion leaders (presumably in the medical and scientific fields) to understand the mechanism of olmesartan and its effects on patients. Ghazzi Dep. 152: 11-16. The only rational inference to be drawn from this evidence is that defendants' employees do not fully understand the cause of SLE. This excerpt cannot substitute for *Daubert*-reliable testimony as to general causation.

Exhibit 9: 2 page excerpt (out of at least 290 pages) from the deposition of Oliseyenum MacDonald Nwose, M.D., defendants' head of medical affairs and "responsible for the Olmesartan drugs", according to plaintiffs' letter request.
Plaintiffs' request does not contextualize the excerpt within the deposition as a whole.

In response to the question whether it is more likely than not that olmesartan causes SLE and serious gastrointestinal problems in some patients, Dr. Nwose states there have been cases "where olmesartan has been associated with sprue-like enteropathy" (Nwose Dep. 289:8-14; 290:1-2) and adds that before forming a conclusion as to causation, he would have to "go back and review each of these cases individually". *Id.* at 290: 7-9.

Here, a possible medical expert eschews assigning the label of causation onto olmesartan for SLE symptoms until he has reviewed each case himself. On its face, Dr. Nwose's testimony is no substitute for expert witness testimony.

Exhibit 10. 1 page excerpt (out of at least 151 pages) from the deposition of Anthony Corrado, Defendants' Director of Commercial Regulatory Affairs from 2011 to 2015.
***7** Plaintiffs' request does not contextualize this excerpt within the deposition as a whole.

Mr. Corrado answers "there is a probability" to the question whether defendants agree that some patients do suffer severe gastrointestinal side effects from taking olmesartan-containing drugs. Corrado Dep. 151: 10-15. Mr. Corrado's answer does not resolve the issue of general causation because there is no elimination of other agents possibly causing SLE symptoms in olmesartan patients. This exhibit is not clear, unambiguous, concrete or sufficient to demonstrate general causation.

Exhibit 11: 4 page excerpt (out of at least 147 pages) from the deposition of Diane Benezra-Kurshan, M.D.
Plaintiffs' request does not contextualize this excerpt within the deposition as a whole and appears to identify Dr. Benezra-Kurshan as that member of defendants' Label Review Committee who drafted proposed warning language to physicians regarding olmesartan use. The date of the proposed warning language is unidentified but after the publication of the Rubio-Tapio article, *supra.*

Dr. Benezra-Kurshan is asked about the meaning of her proposed drug label and its message to physicians. She answers that the label would read to physicians that olmesartan is probably causing the SLE and advises physicians to stop drug ingestion and the SLE symptoms may go away. Benezra-Kurshan Dep. 133:1-8. She adds that the proposed label would also indicate to physicians that, when the drug is stopped, and patients don't improve, then causes other than olmesartan ingestion should be investigated. *Id.* at 147:9-14.

Although this excerpt speaks to defendants' knowledge and response, after the Rubio-Tapio article, *supra*, to the occurrence of SLE symptoms in relation to olmesartan ingestion, it does not suffice as clear, unambiguous, and concrete demonstration of general causation.

Exhibit 12: 12 page excerpt (out of at least 178 pages) from the deposition of Makoto Mizuno, defendants' employee in Japan, *who collaborated on the development of olmesartan.* Mizuno san is asked: "Based on everything you've seen and the study that you were doing in your company with a team of people, you do agree that there is some number of people–we don't have to argue about how many—some people who do develop sprue-like enteropathy from taking olmesartan, correct?" Mizuno Dep. 177: 23 to 178: 5. He responds: "I think that some patients–among some patients who were taking olmesartan, there were some patients who developed sprue-like enteropathy". *Id.* at 178:8-12.

It is unclear why plaintiffs have proffered this exhibit as evidence of general causation since Mizuno san simply states there is a co-occurrence in some patients taking olmesartan with their experience of SLE. The exhibit is not clear, unambiguous, and specific evidence sufficient to demonstrate general causation.

Exhibit 13: 27 page excerpt (out of at least 364 pages) from the deposition of Jeffrey Warmke, Ph.D., defendants' witness under Federal Rule of Civil Procedure 30(b)(6). Dr. Warmke attests that defendants received reports of the occurrence of villous atrophy, and/or gastroenteritis, or collagenous colitis—symptoms complained of in this litigation—in three patients participating in their clinical ROADMAP studies.[11] Warmke Dep. 327:21 to 331:3; 340:18 to 345:18; 348:6 to 349:1. He also attests that defendants' analysts documented that two of these occurrences had a causal relationship to the olmesartan ingestion. *Id.* at 345:6 to 19; 349:13 to 350:10.

***8** Although this exhibit may support specific causation if the patients Dr. Warmke discussed are also plaintiffs in this matter, it does not resolve the issue of the general causation of injuries complained of by all plaintiffs here. *See generally* RESTATEMENT (THIRD) OF TORTS: LIAB. FOR PHYSICAL & EMOTIONAL HARM § 28 comment c (2010) ("The concepts of general causation and specific causation are widely accepted among courts confronting causation issues with toxic agents.").

Exhibit 14: 7 page excerpt (out of at least 137 pages) from the deposition of Dr. Katsuyoshi Chiba, defendants' employee in Japan.

Plaintiffs' request provides no specific reason for including Dr. Chiba's testimony or contextualizes this excerpt within the deposition as a whole nor identifies Dr. Chiba's role for defendants.
Particularly salient in Dr. Chiba's testimony in this exhibit are:

-"it is not possible to reproduce the results of the clinical studies by Mayo Clinic" (Dr. Chiba Deposition Transcript 59:7-8);

-"I think the best scenario would be that this will not be conducted by the non-clinical side" (referring to a non-clinical comparison test of olmesartan with other anti-hypertension drugs that also rely on the action of a TGF-# inhibitor, designed to determine whether all such hypertension drugs are linked to symptoms that plaintiffs complained of) (*Id.* at 64: 21-24);

-" it wasn't a matter of proving or not [the relationship between olmesartan and SLE] but rather it was not possible for us to carry out any kind of nonclinical studies with — with—in — in a reliable manner" (*Id.* at 65: 16-19).

This exhibit appears to relate to whether defendants' choice not to conduct non-clinical tests indicated a belief that such tests would show a causal connection between olmesartan ingestion and SLE symptoms. Dr. Chiba's testimony confirms that, since such a test was not conducted, there can be no information pointing to general causation and therefore cannot demonstrate it.

Conclusion

None of the exhibits proffered by plaintiffs either singly or in combination evidences in a clear, unambiguous, and concrete way the mechanism by which the olmesartan-containing drugs at issue may generally cause the complained of injuries. No exhibit or combination can resolve the inevitable jury speculation as to the complex biochemical, biological, and epidemiological information that underpins the general causation question here.

Consequently, this court declines to find or characterize whether any of the proffered exhibits is an admission by defendants under Federal Rule of Evidence 801(d) (2).

Accordingly, for all the reasons discussed above, plaintiff's Request to File Summary Judgement Motion on Submitted Exhibits has been DENIED in Doc. No. 938.

Dated: 11/8/2016.

All Citations

Slip Copy, 2016 WL 6652358

Footnotes

1 These drugs are Benicar®, BenicarHCT®, Azor®, and Tribenzor®; they are collectively referred to herein as "olmesartan".

2 The Court will collectively refer to all the Daiichi party defendants as "Daiichi."

3 The Court granted plaintiffs leave to take some additional depositions after September 30, 2016, but cautioned this would not extend any other scheduling deadline. See September 1, 2016 Order at 3. [Doc. No. 874].

4 In addition to the cases in this MDL, approximately 73 related cases are consolidated in New Jersey State Court as Multicounty Litigation ("MCL"). Discovery in the federal MDL and state MCL has been coordinated. The Court anticipates a joint Daubert-type hearing will be held in the spring or summer of 2017. The state equivalent to Daubert is *Kemp ex rel. Wright v. State*, 174 N.J. 412 (2002).

5 A. Rubio-Tapio *et al.*, *Severe Spruelike Enteropathy Associated with Olmesartan*, MAYO CLIN. PROC. 87(8), 732:738 (2012).

6 A MedWatch report is a voluntary report to the U.S. Federal Drug Administration (FDA) of an adverse event or undesirable effect associated with using a medical product, including pharmaceuticals and medical devices. The report can be prepared on a one-page FDA form or done via the telephone by health care professionals, patients, and consumers.

7 From a regulatory perspective and in relation to the periodic safety reports (titled in the U.S. as Development Safety Update Report ("DSURs")) provided to a national regulatory agency by the manufacturer of a drug either under development or that has been marketed and under further study, the term "expectedness" relates to whether a physiological reaction is a statistically expected side effect of the pharmaceutical. *Guidance for Industry. E2F Development Safety Report*, U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES. FOOD AND DRUG ADMINISTRATION , prepared by INTERNATIONAL CONFERENCE ON HARMONISATION OF TECHNICAL REQUIREMENTS FOR REGISTRATION OF PHARMACEUTICALS FOR HUMAN USE (ICH), August 2011 (containing nonbinding recommendations), http://www.fda.gov/downloads/Drugs/.../Guidances/ucm073109.pdf.

When categorized as expected, a physiological reaction to drug ingestion must be clearly listed in the Reference Safety Information (RSI) of the DSUR provided by the drug manufacturer to the regulatory agency. *Id.* at 11, 14 and 23.

8 Defendants' unit that does pharmaceutical research, development, and marketing primarily in the U.S.

9 Developed by the International Conference on Harmonisation of Technical Requirements for Registration of Pharmaceuticals for Human Use (ICH), the Medical Dictionary for Regulatory Activities ("MedDRA") provides a globally standardized terminology to regulatory agencies (including the U.S. Food and Drug Administration, the European Medical Agency, and the Japanese Pharmaceutical and Medical Device Agency), pharmaceutical companies, clinicians, and translators, http://www.medra.org.

10 Olmetec® is a trademark registered in Japan to Daiichi Sankyo. Alteis™ is a brand name used by Daiichi Sankyo. These marks identify an olmesartan formulation equivalent to Benicar® and are used to market that in France. http://mpkb.org/home/mp/olmesartan/buying

11 Defendants conducted their own clinical studies of the olmesartan-containing drugs, which were designed to determine a reduction in the level of albumin in a patient's urine. Inasmuch as such albumin is a biochemical indicator of kidney disease due to hypertension, Defendants' ROADMAP tests were to some extent analyzing the statistical effectiveness of olmesartan ingestion on hypertension.

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